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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 41 JANUARY 1945 NUMBER 1

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LEMPERT FENESTRA NOV-OVALIS WITH MOBILE STOPPLE

A NEW ADVANCE IN THE SURGICAL TREATMENT FOR CLINICAL OTOSCLEROSIS EVOLVED AS A RESULT OF A RESEARCH STUDY OF ONE THOUSAND CASES IN WHICH FENESTRATION HAS BEEN PERFORMED DURING THE LAST SEVEN YEARS

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NEW YORK

"Otosclerosis" is the name employed to describe a lesion observed histologically within the bony labyrinthine capsule and also a form of deafness which this lesion occasionally gives rise to before histologic evidence of the presence of the otosclerotic process is obtainable. The lesion known as otosclerosis, which consists of circumscribed, sharply defined, inlaid bony tumors within the otic capsule, has been found during routine histologic examination of the temporal bones of persons who were never known to be deafened, within one or more of the following regions of the otic capsule: the oval window, the round window, the cochlear capsule and the fundus of the internal auditory meatus. The site of predilection for this lesion, however, has been observed to be the region of the oval window, although on rare occasions this region was free of it while other areas of the otic capsule were involved. Whenever the otosclerotic patches in a histologic specimen were observed to have remained within the bony capsule of the labyrinth, no clinical signs or symptoms which could have suggested such a lesion were known to have been present during life. When histologically the otosclerotic tumor was observed to have proliferated beyond either the periosteal or the endosteal layer of the bony capsule in the region of the oval window and to have caused ankylosis of the stapedial footplate, a history of deafness during life was obtainable. This deafness always was of a definite clinical form which is characteristic of and identifiable with the lesion of otosclerosis; the presence of this lesion could be reasonably suspected in a patient presenting the clinical syndrome, and its presence as a rule should be confirmed histologically. However, from the scientific standpoint, I believe that the otologist has no right to refer to this form of deafness as otosclerosis until or unless he has obtained histologic proof of the presence of the sclerotic lesion. Since, however, this type of proof is as yet not feasible in the case of the living patient and since the fenestration of the vestibular labyrinth has been developed as a cure for this form of deafness and is here to stay, I believe that deafness suspected to be due to otosclerosis should be referred to not as otosclerosis but as clinical otosclerosis, which at once signifies that the deafness is suggestive but not positive evidence of the presence of otosclerosis.

Such nomenclature I believe would help greatly to clarify the raison d'etre of the surgical treatment of clinical otosclerosis, make the rationale of fenestration more apparent and its accomplishments as a therapy more easily understood.

Fenestration does not pretend to cure the lesion of otosclerosis in the region of the oval window but can and does cure clinical otosclerosis by rendering the lesion symptom free despite the fact that the suspected otosclerosis may remain and even continue to progress.

BASED ON WHAT SURGICAL PRINCIPLE CAN FENESTRATION OF THE VESTIBULAR LABYRINTH CURE CLINICAL OTOSCLEROSIS?

Clinical otosclerosis can be cured by ignoring the otosclerotic tumor in the region of the oval window, which immobilized the stapedial footplate, and creating instead a new oval window in the surgical dome of the vestibule, which is not a site of predilection for otosclerosis and in which histologically otosclerotic foci have as yet never been observed. Such a newly created window in the bony capsule of the vestibular labyrinth replaces the functionally impeded oval window and establishes a new air conduction mechanism which permits the perilymph and the endolymph to be freely mobilized again by airborne sound. Clinical otosclerosis is thus converted into symptom-free otosclerosis.

HOW CAN A PREOPERATIVE DIAGNOSIS OF CLINICAL OTOSCLEROSIS BE ARRIVED AT?

Whereas the pathologic diagnosis of the lesion of otosclerosis is possible only post mortem, the diagnosis of stapedial ankylosis suggestive of

Read before the Otosclerosis Study Group at the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago, Oct. 8, 1944.

such a lesion, namely, of clinical otosclerosis, can, as a rule, be made in vivo. Grossly, stapedial ankylosis was confirmed on the operating table in about 95 per cent of the last 700 cases in which a preoperative diagnosis of clinical otosclerosis was made. Histologically, stapedial ankylosis due to otosclerosis was confirmed in the single case which came to autopsy postoperatively.

The diagnosis of clinical otosclerosis is simple and is easily arrived at when all or most of the following classic signs and symptoms are observed in a patient complaining of deafness:

1. A family history of hereditary deafness is obtainable.

2. Deafness for air-borne sound is present which is slowly or rapidly progressive in degree and extent, beginning in the lower frequencies first, and ultimately involving the higher frequencies. Such deafness is usually bilateral, though it may appear in one ear first and follow in the other ear some time later.

3. Hearing for bone-conducted sound is prolonged over and above the hearing by air conduction in all the frequencies in which the hearing by air conduction is impaired.

4. The patients frequently believe that they hear conversation better in noisy places.

5. Audiometric testing following inflation of the eustachian tube will show that such inflation does not improve their hearing temporarily, though some patients will claim that it does.

6. They frequently complain of either unilateral or bilateral tinnitus, which may be mild or severe, continuous or intermittent.

7. The tympanic membranes in such patients are usually atrophic and extremely translucent. All the anatomic structures of the middle ear are clearly visible, and the mucous membrane lining the promontory of the cochlea often presents a congested pinkish hue.

8. The eustachian tubes are patent as a rule.

9. Weber test: When there is a marked difference in hearing by air conduction in the two ears, bone-conducted sound is lateralized to the worse of the two ears, provided the hearing by bone conduction in that ear is greater than in the less deafened ear. When the hearing by bone conduction is equal in both, the bone-conducted sound does not lateralize.

10. Hearing for bone-conducted sound may remain normal though the hearing for air-conducted sound may have sunk to a low decibel level, even when the hearing loss reaches as low as the 100 decibel level for all the frequencies.

The clinical picture described in the foregoing ten points on which the diagnosis of the clinical otosclerosis is based and reached is the classic picture. This picture, however, is not encountered at the time of examination in a large percentage of patients deafened from otosclerosis. The otologist must be able to diagnose stapedial ankylosis due to otosclerosis in a deafened person and will often be called on to do so even when the clinical picture that should be presented is masked and obscured.

The diagnosis of clinical otosclerosis cannot be excluded and may be reached in such cases as the following:

1. The patient is unable to furnish a history of familial deafness and even the definite absence of hereditary deafness can be established. The pathologic process of otosclerosis may well have existed and may still exist in the bony labyrinthine capsule of some of the other members of the family without having caused stapedial ankylosis, therefore remaining symptom free.

2. The progressive hearing loss for air-borne sound is unilateral and not bilateral. Since clinical otosclerosis, which is as a rule bilateral, usually manifests itself in one ear first, and several years may pass before clinical manifestations of deafness in the other ear appear, the diagnosis of clinical otosclerosis in a patient presenting himself to the otologist before his second ear has become involved cannot be ruled out on this basis alone.

3. The observation of the apparent ability to hear conversation better in noisy than in quiet surroundings has not been made by the patient. Some patients are less observing than others.

4. In the Weber test hearing of bone-conducted sound is not lateralized to the ear with the worse hearing for air conduction despite the fact that the hearing by bone conduction has remained good in the ear with the worse hearing for air-conducted sound. This test is often not reliable because such factors as tinnitus and the patient's inability to concentrate are important in determining its accuracy.

5. One or both tympanic membranes are found not to be atrophic and translucent but scarred, thickened and cloudy at the time of examination, or calcareous deposits or weblike healed perforations are seen in one or both. A diagnosis of clinical otosclerosis cannot be excluded on the basis of these findings alone. There is no reason why a patient having clinical otosclerosis cannot contract unilateral or bilateral otitis media which resolves, leaving behind any of the described changes within the tympanic membrane without having caused serious damage to the air conduction mechanism. By the same token there is no reason why a patient in whom during his childhood unilateral or bilateral otitis media occurred without damaging at all or damaging only slightly the air conduction mechanism outside of leaving the described changes in the tympanic membrane could not eventually in adult life have contracted clinical otosclerosis which when superimposed on the already partially damaged air conduction apparatus resulted in the enhanced degree of deafness now encountered.

6. In tests of bone conduction, abnormalities suggestive of progressive degeneration of the cochlear nerve secondary to stapedial ankylosis of otosclerotic origin are encountered; the hearing for bone-conducted sound is prolonged over and above the below-useful level of air conduction hearing in the lower frequencies, while in the higher frequencies both the air conduction and the bone conduction hearing are lower than in the low frequencies or even totally gone. In such a case the diagnosis of clinical otosclerosis cannot be excluded.

7. The patient's history and the clinical and physical findings point to the presence of a mixed form of deafness involving both the air conduction and the hearing perception mechanisms in different degree and independently, the changes in the two mechanisms being not related etiologically to each other: A certain amount of the deafness resulted from damage to the organ of Corti as a consequence of a systemic infection or of acoustic trauma, and a certain amount resulted from stapedial

ation due to otosclerosis. The cochlear nerve which is damaged, however, is still capable of greater function than it is called on to supply by the functionally impeded air conduction mechanism. Whether the damage to the organ of Corti following toxic neuritis took place prior to or after the onset of the air conduction deafness due to stapedial ankylosis is inconsequential. As judged by the clinical findings in a case of mixed middle and inner ear deafness, the middle ear deafness resulting from the stapedial fixation seems to be the predominating form of deafness and responsible for the major degree of the mixed deafness, a diagnosis of clinical otosclerosis predominating in a mixed form of deafness may be reached.

8. Deafness for air-conducted sound of the type encountered in otosclerosis is present in one ear and nerve deafness for perception of sound due to damage of the organ of Corti as a result of toxemia is present in the other ear. A diagnosis of unilateral clinical otosclerosis can be reached in the presence of these findings.

9. A dead labyrinth as a result of acute serous or suppurative labyrinthitis following an acute infection of the middle ear is present in one ear while a progressive form of air conduction deafness with prolonged bone conduction is present in the other ear in which an infection of the middle ear has never occurred. In such a case a diagnosis of unilateral clinical otosclerosis can be made.

WHEN IS DEAFNESS DIAGNOSED AS CLINICAL OTOSCLEROSIS SUITABLE FOR FENESTRATION?

Since deafness resulting from otosclerosis is progressive and usually results in total irreparable loss of hearing due to secondary degeneration of the organ of Corti, and since there is no known satisfactory medical treatment which would either improve the hearing or arrest the process, and since both experience and time have shown that in a large percentage of patients this form of deafness can be cured only by surgical treatment of the labyrinth, fenestration is indicated in any of the following cases:

1. Hearing for normally spoken conversation has dropped to a level where it is no longer useful, serviceable or practical for everyday social and economic contacts. When the hearing for air-borne sound as tested audiometrically drops below the 30 decibel level in the key conversational frequencies, it ceases to be of practical value and becomes less useful as it keeps dropping to a still lower decibel level.

2. There is not at the time of operation an active acute, subacute or chronic suppuration of the middle ear. A previously occurring healed infection of the middle ear is no contraindication.

3. The drum prior to operation is intact and not perforated. A scarred drum, a healed perforation, a calcified, thickened, hazy or retracted drum is no contraindication.

4. There is lack of positive evidence that the membrane of the round window is functionally impeded.

5. There is reason to suspect that there still exists in the ear chosen for operation a reservoir of cochlear nerve function in excess of what is being utilized or tapped by the functionally impeded air conduction mechanism and that this reservoir is sufficient to permit improvement of hearing as a result of improving the air conduction mechanism.

HOW CAN THE RESERVOIR OF COCHLEAR NERVE FUNCTION BE DETERMINED?

When I first began to treat clinical otosclerosis surgically, I firmly believed that hearing for bone-conducted sound as determined with the audiometer or tuning forks was the infallible index of cochlear nerve function. I therefore stated in 1938,[1] 1939[2] and 1940[3] that fenestration of the labyrinth for the improvement of hearing in clinical otosclerosis was indicated only when the hearing by bone conduction for the frequencies 512, 1024 and 2048 as determined audiometrically under the condition of masking has remained normal or receded to a level not lower than 30 decibels. After having operated on 375 patients and, among them, a considerable number who requested and insisted on being treated by fenestration, despite the fact that their audiometrically determined hearing for bone conducted sound fell short of the required minimum which I considered essential for obtaining a successful end result, I was surprised to find that in many of them the hearing loss was greatly lessened and in about 30 per cent of the cases which I believed to be nonsuitable because of the poor bone conduction demonstrated, the hearing was restored to the practical, serviceable level as a result of the fenestration operation.

Therefore, in 1941[4] I no longer stated that fenestration of the labyrinth is indicated when the hearing by bone conduction has not yet receded to a level lower than 30 decibels for the conversational frequencies but stated instead that fenestration of the labyrinth is indicated when the impairment of hearing in the 512, 1024 and 2048 frequencies is not the result of degeneration of the cochlear nerve.

Today, as a result of my experience with the fenestration operation in 1,000 cases, I believe that the significance of the amount of bone conduction hearing for individual sound frequencies found in a particular case with the aid of the audiometer or tuning forks and its relation to

1. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis: A New One-Stage Surgical Technic, Arch. Otolaryng. **28**:42-97 (July) 1938.

2. Lempert, J.: Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis: Summary Report of 120 Cases, Arch. Otolaryng. **31**:711-779 (May) 1940.

3. Lempert, J.: Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis: Indications, Technique, Observations as to Early and Late Postoperative Results, Laryngoscope **51**:330 (April) 1941.

4. Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880-912 (Nov.) 1941.

the degree of cochlear nerve function is but little understood.

I observed that in a substantial number of cases in which one, two or even all three of the conversational frequencies were heard by bone conduction at a level lower than 30 decibels preoperatively the hearing by air conduction was greatly improved following fenestration and even restored to the practical, serviceable level and in some to normal (fig. 1 *A* and *B*).

By the same token of experience I found that in those cases of clinical otosclerosis in which the involved ear was operated on successfully in spite of the observation that preoperative hearing by bone conduction was much below the 30 decibel level in one, two or even three conversational frequencies and in which the hearing by air conduction was greatly improved, the hearing for bone-conducted sound in these frequencies was also improved. The fact that hearing by bone conduction could ever be improved by fenestration of the labyrinth would suggest either that such hearing cannot always be accurately determined with present methods of testing or that bone conduction hearing and cochlear nerve function potential are not synonymous (fig. 1 *C*).

If the presence and the degree of bone conduction hearing are to be employed as an absolute barometer of the presence and the degree of cochlear nerve function, a person with no discernible hearing for bone-conducted sound should be totally deaf. However, this is often not the case; one may have a total loss of hearing for bone-conducted sound and yet hear by air conduction the normally spoken voice for a distance of 1 to 5 feet (0.3 to 1.5 meters) (fig. 2).

I have seen patients with clinical otosclerosis who on audiometric testing showed a loss of hearing for air-borne sound not reaching lower than the 35 or 40 decibel level for all the frequencies and who had normal hearing by bone conduction and yet were unable to hear the normally spoken voice for a distance of more than 2 to 3 feet (0.6 to 0.9 meter). By the same token, I have encountered patients with clinical otosclerosis who on audiometric testing showed a loss of hearing for air-conducted sound reaching as low as the 60 decibel level for all the frequencies and who were able to hear the normally spoken voice for a distance of 8 to 10 feet (2.5 to 3 meters). Since no discernible difference in bone conduction hearing could be found in these patients, and if their hearing by bone conduction represented the degree of cochlear nerve function, which must be assumed to be equal in their cases, it is difficult to understand why an ear in which the hearing loss for air-borne frequencies has dropped to the 60 decibel level should ever hear the spoken voice better than an ear in which the hearing by air conduction for sound frequencies has dropped only to the 35 or 40 decibel level.

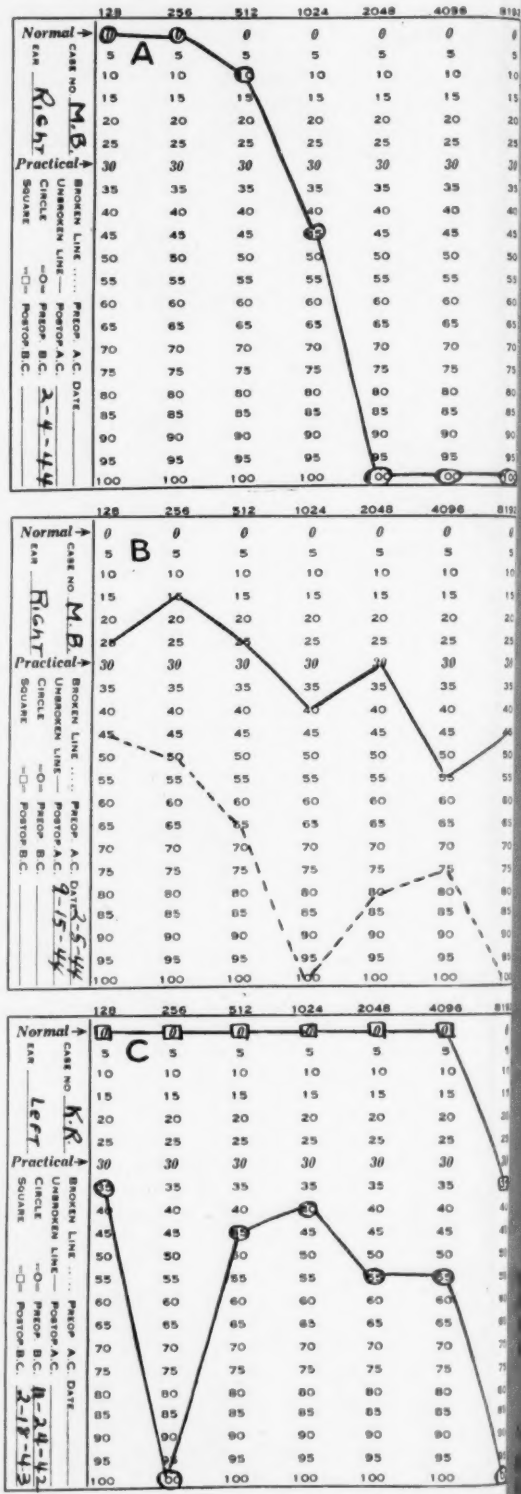

Fig. 1.—*A*, right ear of M. B.; preoperative hear for bone conduction was below the 30 decibel le *B*, right ear of the same patient; postoperative impro ment in hearing despite poor bone conduction. *C*, ear of K. R.; poor preoperative hearing for bone c duction restored to normal postoperatively.

Such experience and observations led me to conclude: first, that the accurate determination of the decibel level of bone conduction hearing is not always possible; second, that the most accurately determined decibel level of bone conduction hearing for individual sound frequencies is not always representative of the degree of cochlear nerve function still present in a deafened ear.

inaccuracies accompanying such tests, is employed to measure the cochlear nerve function still present.

The findings from such tests of bone conduction should be studied in each case and evaluated in conjunction with the rest of the clinical picture of deafness. The decision as to the possibility of improving the hearing by fenestration in a particular ear should be arrived at only after

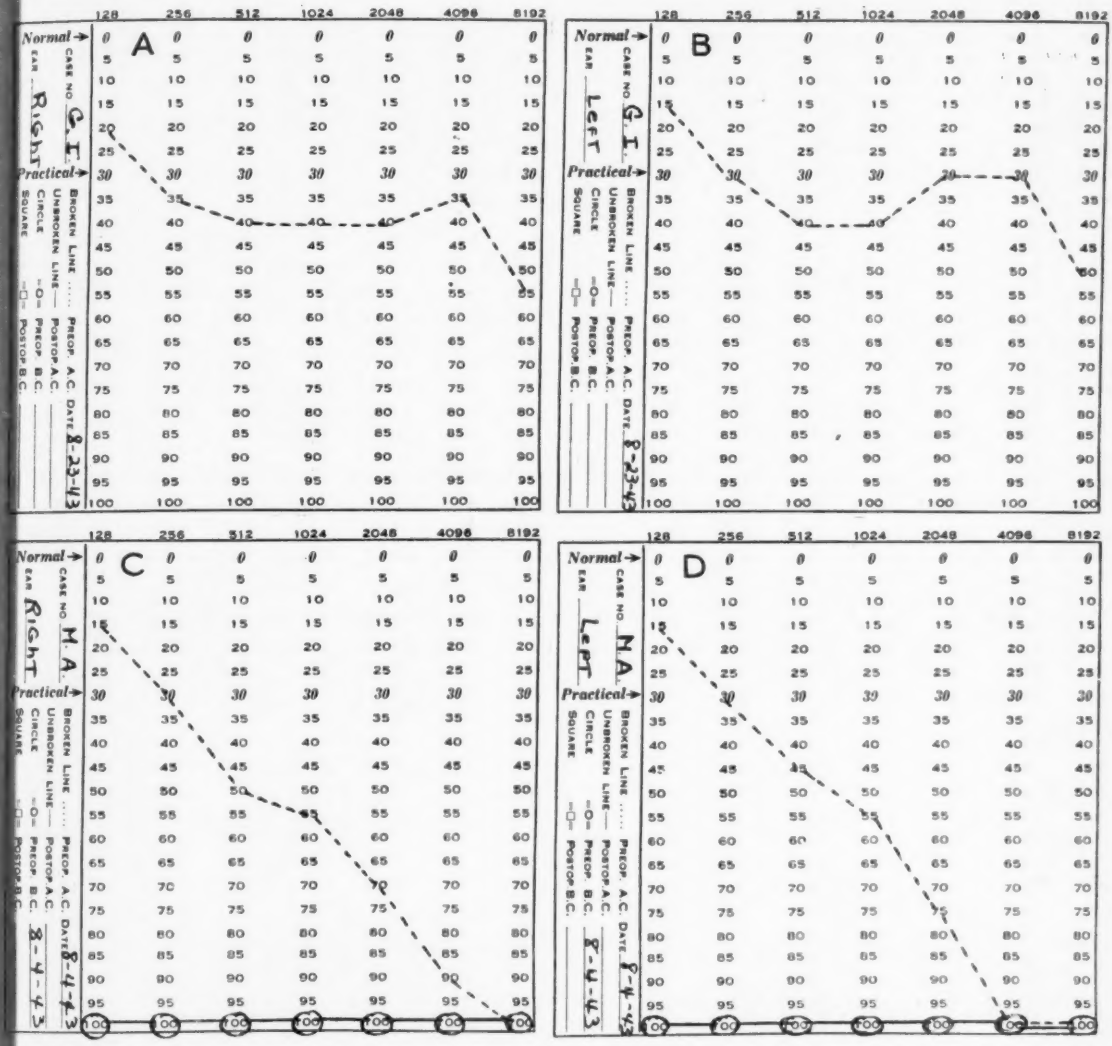

Fig. 2.—*A* and *B*, right and left ears of G. I.; complete absence of hearing by bone conduction for any of the tuning forks in each ear. The patient heard the normally spoken voice for a distance of 5 feet (1.5 meters) in each ear. *C* and *D*, right and left ears of M. A.; complete absence of hearing by bone conduction as tested with tuning forks and an audiometer. The patient heard the normally spoken voice in each ear for a distance of 1 foot (0.3 meter).

One can readily see that in view of such observations and conclusions no hard and fast general rule as to the requirements of hearing by bone conduction for the determination of the operability of a particular case of clinical otosclerosis for the fenestration operation can be applied as long as the hearing by bone conduction of individual sound frequencies as tested with the audiometer or tuning forks, with all the

taking into consideration the clinical picture as a whole and not any individual symptom or sign.

An accurate, infallible test for the determination of the reserve of cochlear nerve function within an ear deafened to air conduction in clinical otosclerosis is yet to be developed.

A Proposed Test.—There are cases of advanced clinical otosclerosis with total loss of hearing for air-borne sound in both ears. The

patients cannot hear the spoken voice, no matter how loud, in either ear. When tested audiometrically or with tuning forks they show complete absence of hearing for both air-borne and bone-conducted sound.

When tested with an air conduction hearing aid they show no response to the spoken voice but hear and respond to the spoken voice when tested with a bone conduction hearing aid (fig. 3).

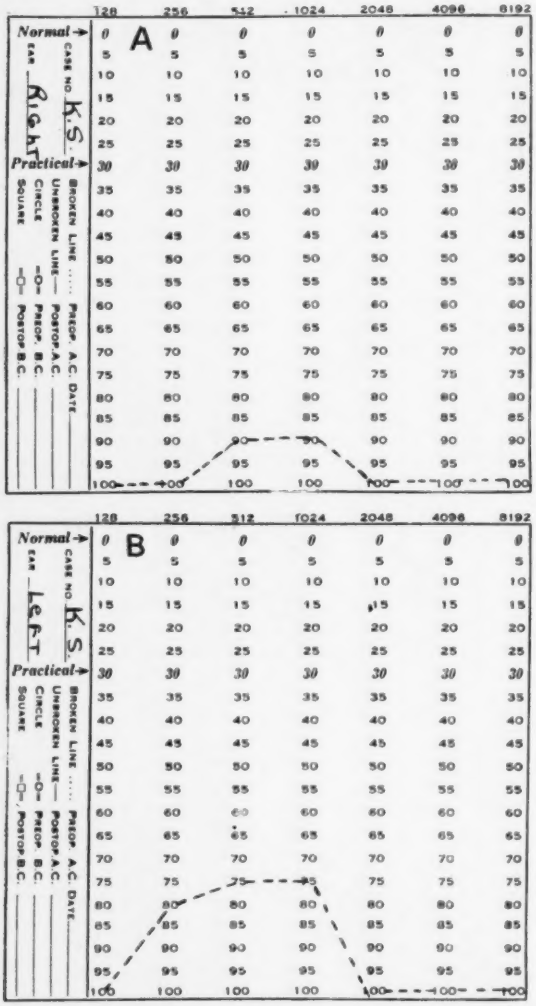

Fig. 3.—*A*, right ear of K. S.; no hearing by air conduction or bone conduction for either the spoken voice or pure audiometric tone; no hearing with either an air or a bone conduction hearing aid. *B*, left ear of the same patient; no hearing for the spoken voice no matter how loud; complete absence of hearing by air conduction and by bone conduction audiometrically; no hearing for the tuning forks either by air or by bone conduction; no hearing with an air conduction hearing aid. The patient did hear with a bone conduction hearing aid for a distance of 1 foot (0.3 meter).

Such cases clearly demonstrate that a person who is apparently totally deaf for the ordinarily spoken voice and for both air-borne and bone-conducted sound may still possess some cochlear nerve function, the degree, the quality and t quantity of which are not determinable by te at present available.

Such cases show that with the sound frequen tests used the presence or the absence of heari function is not always determinable and that t degree, the quality and the quantity of the p tential of cochlear nerve function which is n being tapped by the functionally impeded a conduction mechanism in a case of staped ankylosis and which could be brought into u by fenestration cannot be estimated accurat by present methods of testing.

A Suggestion.—The following test for t determination of the degree of cochlear ner function still in reserve in a case of air co duction deafness is suggested:

An audiometer for testing speech by air co duction and bone conduction instead of sou frequencies should be constructed. The patie should be asked to repeat a series of wor recorded on a disk which contain in the aggr gate all the vowels and consonants in the alph bet by listening to the speech audiometer throu an air conduction receiver, and the decibel le at which he no longer hears the spoken wor should be noted. The air conduction receiv should then be replaced by a bone conducti receiver, and if the patient resumes hearing t recorded spoken voice with this receiver at t decibel level where he ceased hearing with t air conduction receiver, the audibility is grad ally lowered to the decibel threshold where t recorded speech is no longer heard by bone co duction.

By measuring the bone conduction hearing f recorded speech between the decibel level which the recorded speech can no longer heard by air conduction and the decibel level which the recorded speech ceases to be heard bone conduction, reliable information should obtained as to the degree of cochlear nerve fun tion reserved in a deafened ear.

AT WHAT STAGE OF THE LOSS OF HEARING F AIR-CONDUCTED FREQUENCIES SHALL ONE OPERATE IN ORDER TO EXPECT RESTORATION OF SERVICEABLE HEARING?

Whether fenestration results in improved, p tially serviceable hearing or in restoration practical, fully serviceable hearing is not dep dent on the degree of air conduction hearing s observable at the time of operation but rat on the amount of cochlear nerve function rema ing untapped by the functionally impeded conduction mechanism. No patient with clin otosclerosis should as a rule be advised to s ject himself to the operation before his loss

aring by air conduction has reached at least e 40 decibel level for most of the conversa- nal frequencies, at which stage he is deaf for practical purposes. It is not the least bit ore difficult to restore hearing by fenestration an ear with a 70 to 80 decibel loss of hearing the 30 decibel level which is necessary for actical, serviceable hearing, or even to a higher vel, than it is to restore hearing to that same gh level postoperatively in an ear with a 40

WHEN BOTH EARS ARE SUITABLE FOR FENESTRATION, WHICH OF THE TWO DEAFENED EARS SHOULD BE CHOSEN FOR OPERATION?

The ear which most likely can be expected to yield the type of hearing improvement which will be most beneficial to the patient should be the one chosen for operation. When both ears are deafened to the degree that practical hearing no longer exists in either ear, the ear suspected to possess the greater reservoir of coch-

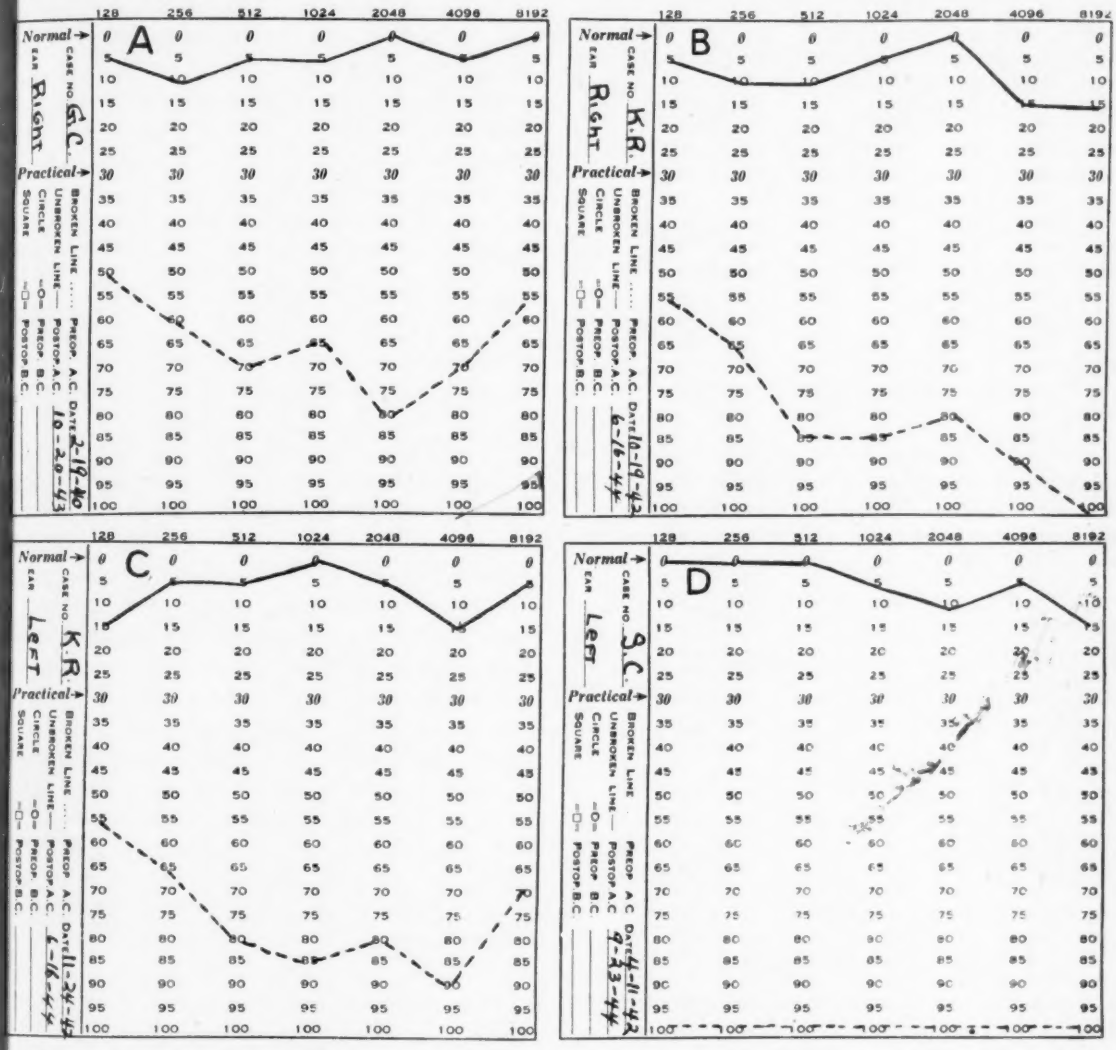

Fig. 4.—*A*, right ear of G. C.; hearing restored to normal. *B* and *C*, right and left ears of K. R.; hearing restored to normal. *D*, left ear of S. C.; hearing by air conduction restored to normal though a total loss was recorded preoperatively.

ecibel loss of hearing, provided that in each stance an untapped adequate reservoir of ochlear nerve function exists (fig. 4 *A*, *B* and).

It was also my privilege to find out that nor- al hearing can be restored to one suffering om total air conduction deafness, with 100 ecibel loss in all the frequencies, when the ochlear nerve function has remained normal ig. 4 *D*).

lear nerve function should be chosen, even if that ear is the better functioning of the two from the standpoint of air conduction hearing. When the reservoirs of cochlear nerve function are suspected to be equal, the ear with the poorer air conduction hearing should be chosen. When the difference in the air conduction hearing is not too great in the two ears, either ear may be chosen, provided there is reason to suspect that the reservoir of cochlear nerve function is about

the same in the two ears. If tinnitus is present in one ear and not in the other, or if the tinnitus is more severe in one ear than in the other, and if there is reason to believe that the cochlear nerve function in the ear with the tinnitus is still good enough for one to expect that hearing could be improved, the ear with the tinnitus or the ear with the severer tinnitus should be chosen if the hearing by air conduction in that ear is below the practical level and even if the hearing by air conduction in the other ear is much worse. The operation results in the abatement of tinnitus in the fenestrated ear in about 95 per cent of the cases in which practical hearing is obtained by operation and for just as long as such hearing is postoperatively maintained.

WHAT SHOULD BE ONE'S OBJECTIVE IN OPERATING FOR CLINICAL OTOSCLEROSIS?

There are two objectives: (1) to restore the air conduction hearing as near to audiometric normalcy as the existing reservoir of untapped perceptive hearing function will permit and to the limits of the functional capacity of the organ of Corti in each case; (2) to maintain continuously and permanently such improved hearing at the highest obtainable level.

CAN THE OPERATION RESTORE PHYSIOLOGIC HEARING TO THE SERVICEABLE LEVEL NECESSARY FOR SOCIAL AND ECONOMIC CONTACTS?

Not until one succeeds in improving the hearing at the 512, 1024 and 2048 frequencies to the 30 decibel level does one succeed in giving the patient practical, serviceable hearing for all social and economic contacts.

On the creation of a new fenestra in the external semicircular canal in 300 ears and in the surgical dome of the vestibule in 700 ears, to take the place in each case of the functionally impeded oval window, the hearing was observed at the operating table to have been immediately restored to the practical level or higher in every case of clinical otosclerosis in which a sufficient reservoir of cochlear nerve function, which had remained untapped by the functionally impeded air conduction mechanism, still was present, no matter how great a loss of hearing for air conducted sound was recorded prior to the fenestration. This is today a well established fact that no one doubts. This is sufficient proof that the principle of creating a new window to restore hearing in certain cases of clinical otosclerosis is physiologically a correct one. However, the physiology involved in and responsible for this restoration of hearing following the creation of a new labyrinthine window is still debatable.

The theory that the newly created oval window replacing a functionally impeded oval window permits the perilymph and the endolymph to be once more freely mobilized by air-borne sound appears to be logical.

CAN HEARING RESTORED BY FENESTRATION TO THE SERVICEABLE LEVEL BE CONTINUOUSLY MAINTAINED THEREAFTER AT THAT LEVEL?

The degree of improvement in air conduction hearing observed at the operating table immediately after fenestration of the vestibular bony labyrinth can be maintained continuously thereafter without subsequent appreciable deterioration provided:

1. Osteogenesis does not close the newly created fenestra in the bony capsule of the vestibular labyrinth.

2. The endolymphatic labyrinth does not undergo postoperative inflammatory changes severe enough to result in some degree of permanent damage to the organ of Corti.

WHAT WAS DONE TO PREVENT CLOSURE OF THE NEWLY CREATED FENESTRA AS A RESULT OF OSTEOGENESIS SO THAT THE HEARING IMPROVEMENT COULD BE CONTINUOUSLY MAINTAINED?

Osteogenesis occurs within the freshly cut bony walls of a newly created fenestra and results in bony closure of the fenestral gap because it is the natural tendency of bone when injured to regenerate. Signs and symptoms of its occurrence may begin to manifest themselves as early as four to six weeks postoperatively or as late as eight to twelve months postoperatively. The hearing in such cases recedes gradually to the preoperative level within a period varying from three months to fifteen months after the operation when the new window is completely closed by new bone.

In my effort to keep the newly created fenestra from closing by bone regeneration I attempted to prevent osteogenesis from taking place in five ways:

1. I tried to intimidate, impede and stop the natural tendency toward osteogenetic repair from its source of origin within the freshly cut bony walls of the fenestra. To accomplish this, an attempt was made to damage and alter the normal histologic character of the bony walls of the fenestra in the following ways:

(*a*) Burnishing: The bony labyrinthine capsule was fenestrated in all the ears operated on with the use of the dull dental plug finishing bur

imal experimentation has since shown that employment of this type of burr results in y of new bone formation but does not prevent occurrence.[5]

(*b*) After having created the fenestra with the dull plug finishing burr in all of the ears operated on the freshly cut bony walls of the fenestra were burnished variously with either a 22 karat solid gold burnishing burr, a steel burnishing burr, a lead burnishing burr or a solid silver burnishing burr, in order to damage still further the osteogenetic properties of these walls. Each type of burnishing burr was employed in a separate series of cases and the end results of each series were studied and compared.

(*c*) In one group of cases I silver-plated the freshly cut bony walls with silver amalgam.

(*d*) In another group of cases I burnished the freshly cut bony walls with the electrocoagulating current, employing especially devised electrocoagulating needles for this purpose.

As a control for the evaluation of the principle of burnishing as a means of impeding bone regeneration, I operated in a series of cases in which the walls of the fenestra were deliberately not burnished at all. Observation and comparison of the end results obtained with and without burnishing strongly favored burnishing of the walls as an added means of preventing closure of the fenestra by osteogenesis.

(*e*) Roentgen Therapy: Spontaneous fractures of the clavicle, the humerus or ribs result from use of high voltage roentgen rays in the treatment of carcinoma of the breast, and such fractures never heal. When the bony walls of labyrinthine capsule are necessarily injured the act of creating the new fenestra, the osteogenetic cells within the freshly cut bony walls of fenestra soon begin to grow and undergo division during growth. Thus osteogenetic cells behave like tumor cells, which also show division during growth. It is therefore logical to assume that osteogenetic cells when growing and repairing bone are more radiosensitive than when quiescent, just as tumor cells are. Armed with this theory plus the observations made in the roentgen treatment of carcinoma of the breast, I attempted in a series of cases to prevent osteogenesis by applying roentgen rays to the freshly cut bony walls of the fenestra.

2. I further attempted to prevent osteogenesis by carefully avoiding and eliminating new bone formation originating and resulting from any source other than the freshly cut walls of the fenestra.

(*a*) Removal of Bone Slivers: With the aid of Zeiss magnifying lenses, the inner margins of the freshly cut bony walls of the fenestra, the endosteum and the perilymph space in the region of the fenestra were always carefully searched for the presence of minute slivers of bone, which are part of the fractured endosteal layer of the bony labyrinthine capsule in the region of the fenestra. When observed, these slivers were immediately and carefully removed. The inner surface of the tympanomeatal membrane, which is destined to cover the region of the fenestra, was always carefully examined with the Zeiss magnifying glasses in a search for minute and extremely thin splinters of bone, which were commonly found to be attached and adherent to it as a result of the removal of the bony walls of the external auditory canal. Such splinters when observed were removed, care being taken not to perforate the tympanomeatal membrane. Splinters of bone when unobserved and not removed will rapidly cause closure of the new fenestra by bone regeneration—much more rapidly than when the regeneration of bone originates solely from the freshly cut bony walls of the fenestra. Closure will be complete within a period of two to three weeks postoperatively. Such bone splinters have retained all the histologic structure and properties essential to osteogenesis, and therefore when in contact with the freshly cut bony walls of the fenestra they act like a bone graft, with osteogenesis taking place both from the walls of the fenestra and from the splinters.

(*b*) Removal of Bone Sand: Bone sand is a by-product which results from creating the fenestra with the dull plug finishing burr and should be removed like any other debris in the nettoyage of the surgical wound. Removal of bone sand, which in the process of its creation has lost all its histologic bone structure and therefore all its power for bone regeneration, will not prevent bone regeneration from taking place in the freshly cut bony walls of the fenestra. Holmgren, who made the fenestra with a sharp chisel without creating bone sand, reported that in most of his cases the fenestra closed, which proves that the original or the subsequent elimination of bone sand from the region of the fenestra will in itself not prevent closure of the fenestra by bone regeneration.

Bone sand in the region of the fenestra is best removed by wiping it away with cotton applicators soaked in saline solution, because the sand has a tendency to stick to the surface of the bone

5. Lempert, J.; Meltzer, P. E., and Spector, B.: Bone Repair Following Defects Made in the Skull of Guinea Pigs by Means of Different Instruments, Arch. Otolaryng. **36**:473-490 (Oct.) 1942.

or the endosteum in the form of a thin paste. There is no danger of bone sand entering and accumulating in the perilymph space because all the destruction of bone essential to the creation of the fenestra when accomplished with the plug finishing burr is completed before one removes the endosteum to expose the perilymph space. All the bone sand which is thus deposited on the presenting surface of the endosteum is automatically removed by removing the endosteum.

3. I further tried to prevent osteogenesis by avoiding direct contact of the freshly cut bony walls of the fenestra with any tissue which may stimulate and accelerate osteogenesis.

When covering and protecting a newly created fenestra with the tympanomeatal membrane, I have always tried to place the membrane so that the inner epithelium-lined surface of Shrapnell's membrane would cover and lie in contact with the freshly cut bony walls of the fenestra instead of the periosteum-lined inner surface of the cutaneous lining of the external auditory canal. This is based on my belief that a periosteum-lined surface of a membrane when in contact with a freshly injured bony surface will encourage bone regeneration.

4. I attempted to prevent closure of the newly created fenestra as a result of osteogenesis by increasing the distance that osteogenesis had to travel to bridge the fenestra, hoping that it would exhaust itself before it completed its natural mission of closing the gap.

To accomplish this, I employed in the last 700 cases a new technic whereby the new fenestra was created in the surgical dome of the vestibule. In this region the fenestra can be made much wider in its diameter because the perilymphatic space in the region of the vestibule is much wider than the perilymphatic space in the region of the external semicircular canal. I named this newly created fenestra "fenestra nov-ovalis."

Since the perilymph space in this region is also much deeper than in the region of the external semicircular canal, the formation of a fibrous connective tissue matrix for new bone becomes more difficult.

As a result of this forward shift of the fenestra to the surgical dome of the vestibule, osteogenetic closure of the fenestra has been observed to date less frequently in the 700 cases in which the fenestra nov-ovalis was created than in the first 300 cases, in which the fenestra was created in the external semicircular canal posterior to its ampullated end.

5. I attempted to prevent closure of the new fenestra by physically impeding the pathway for the spread of osteogenesis:

(*a*) Especially devised inert metal obtura frames were introduced into the newly crea fenestras. Each was spread and fitted to line freshly cut bony walls of the fenestra without structing or obliterating the fenestral gap.

Because of the difficulties encountered in making, introducing and continuously mainta ing in position of the inert metal obtura frames, their use had to be abandoned.

(*b*) A tightly fitting solid inert metal turator was inserted into the newly created b fenestra, completely closing and obliterating gap between the fenestral walls, in order physic ly to prevent osteogenesis. After remaining situ for two to three months and thus stiff nature's tendency toward regeneration of b it was removed in the second stage of a stage fenestration operation. The mechan problem of shaping and preparing such a h metal obturator at the time of operation so t it would fit snugly the individually sized shaped fenestra in each case was difficult time consuming.

Comment.—Some critics of this surgical tre ment for clinical otosclerosis who are not quainted with the facts may argue that the re to so many varied technics for the prevention osteogenetic closure of the newly created w dows in the 1,000 ears operated on is a sign failure. This, however, is not true. It is jus sign of my own restlessness resulting from continuous desire to improve the already tainable high percentage of good end results ultimately to perfect a technic which would re in the maximum obtainable of good end resu The percentage of new windows that remai permanently open as a direct result of my evo tionary surgical efforts long since greatly distanced the results of all the previous efforts my predecessors in this field of surgery.

DOES A PERMANENTLY OPEN FENESTRA A WAYS RESULT IN A CONTINUOUSLY MAINTAINED IMPROVEMENT OF HEARING?

Ever since the possibility of improving he ing in clinical otosclerosis by fenestration of vestibular labyrinth was first observed, it been generally believed and claimed by otolog that if the newly created window could be k open permanently, the hearing improvement tained by creating such a window would continuously maintained. My experience w 1,000 fenestrations does not lend support to generalized belief and contention.

I could point to many cases in my series wh preoperatively were most ideally suited for

estration operation and in which the operation was faultlessly done, so that I had the right to expect a good end result but in which, nevertheless, the result brought nothing but disappointment to me and my patient because the remarkable hearing improvement which was apparent for the first few postoperative days soon disappeared, never to return again, despite the fact that the newly created fenestra remained permanently open. Still more distressing for me to behold were those patients who, though ideally suited and faultlessly operated on, with their hearing improved postoperatively, nevertheless not only lost their improvement several days after the operation but sustained a permanent further loss of hearing, reaching a level lower than that experienced preoperatively, despite the fact that the new window remained permanently open.

Careful investigation of the symptoms and signs observed in my patients during the first three postoperative weeks suggested that all such patients undergo postoperatively an inflammation of the endolymphatic labyrinth, symptoms and signs of its existence manifesting themselves usually two to three days postoperatively. In the vast majority of cases this inflammation of the labyrinth completely subsides within a period of ten to twenty-one days after its onset, leaving the endolymphatic labyrinth undamaged. In such cases the hearing improvement obtained immediately following fenestration continues for the first two to three postoperative days and then suddenly subsides and the hearing even drops to a level much lower than that noted preoperatively and stays at that low level for ten to twenty-one days, when it suddenly and rapidly returns to the improved level of hearing obtained immediately after fenestration. In a small percentage of cases the same symptoms and signs of postoperative inflammation of the endolymphatic labyrinth occur except that the recession in hearing which took place two to three days postoperatively remains indefinitely, suggesting that the endolymphatic labyrinth is permanently damaged as a result of the postoperative inflammation which it has undergone. The amount of residual hearing in such an ear varies with the degree of damage sustained by the organ of Corti. Only in too few cases no symptoms or signs of postoperative inflammation of the endolymphatic labyrinth manifest themselves and the postoperative improvement of hearing obtained by fenestration does not recede even temporarily.

Still more significant was the observation that following revision of the newly created fenestra in cases in which closure by new bone formation was suspected, the risk of permanent damage being sustained by the endolymphatic labyrinth as a result of inflammation was greatly enhanced and the percentage of cases in which the hearing was depressed to a level much lower than the preoperative level was greatly increased as compared with the percentage for the primary operation.

The probable cause for this increase is the fact that a membranous labyrinth which has undergone postoperative inflammation following the original operation and which has resisted and escaped permanent damage finds it more difficult to resist and withstand successfully in its already lowered state of resistance a second attack of inflammation following a second operation.

In searching for the cause of the postoperative inflammation of the membranous labyrinth and the factors responsible for variations in the severity of such inflammation, it occurred to me that the tympanomeatal membrane which covers and seals the newly created fenestra and which lies in direct contact and communication with the perilymph space and the endolymphatic labyrinth is most likely the source of the serous labyrinthitis. The unavoidable postoperative inflammation which this membrane undergoes spreads to the perilymph space and extends to the endolymphatic labyrinth. Whether permanent damage to the organ of Corti results from the extension of the inflammation to the perilymph space depends on the severity of the postoperative inflammation in the tympanomeatal membrane and the degree of natural resistance to inflammation existing in the individual endolymphatic labyrinth.

WHAT WAS DONE TO PREVENT POSTOPERATIVE LABYRINTHITIS SO THAT THE HEARING IMPROVEMENT COULD BE CONTINUOUSLY MAINTAINED?

Acting on the supposition that postoperative labyrinthitis with damage therefrom to the organ of Corti is the direct result of inflammation of the tympanomeatal membrane extending into the perilymph space, I attempted, first, to reduce the severity of the unavoidable postoperative inflammation of the tympanomeatal membrane as follows:

1. Prophylactic sulfadiazine therapy was administered for the first five postoperative days.

2. In the last 600 cases the risk of the tympanomeatal membrane undergoing a severe postoperative inflammatory reaction has been considerably reduced by the employment of paraffin instead of paraffin-impregnated mesh gauze to hold the tympanomeatal membrane in position over the newly created fenestra. Melted paraffin was poured into the postoperative wound and

permitted to cover the entire tympanomeatal membrane, including the membrana tensa thereof, until the paraffin reached the level of the outer mastoid cortex. This liquid paraffin hugs the contour of the inner surface of the postoperative wound and solidifies in about ten minutes. It makes contact with the tympanomeatal membrane but does not compress it as paraffin-impregnated mesh gauze does, and therefore it does not interfere with circulation in the membrane. It exerts only a mild uniform pressure in its contact with the membrane, and thus the postoperative inflammation of this membrane is held to a minimum.

Though I succeeded as a result of the aforementioned efforts in reducing appreciably the severity of the postoperative inflammation of the tympanomeatal membrane covering the newly created fenestra, there was no appreciable decrease in either the frequency or the severity of the postoperative labyrinthitis as a result of such treatment of the tympanomeatal membrane.

It thus became obvious that just as long as the newly created fenestra in the bony vestibular capsule had to be kept open and covered only by the tympanomeatal membrane which contacts and communicates directly with the perilymphatic space, the inevitable postoperative inflammation of this membrane even when mildest in degree must naturally extend into the perilymphatic space and involve the endolymphatic labyrinth to a degree proportionate to the degree of inflammation within the tympanomeatal membrane and the degree of natural resistance to inflammation offered by the endolymphatic labyrinth.

Convinced by this time that postoperative labyrinthitis could not be altogether prevented by reducing the postoperative inflammation within the tympanomeatal membrane, I decided to attempt to impede the extension of this inflammation into the perilymphatic space in order to prevent damage to the endolymphatic labyrinth and the organ of Corti as follows:

1. In a series of 100 cases, while the fenestra in the bony capsule of the surgical dome of the vestibule was being created, the shredded and pulverized bony particles of the endosteal surface of the bony labyrinthine capsule which were seen to adhere to the presenting surface of the endosteal perilymphatic membrane were carefully removed, great care being taken not to injure but to leave completely intact the endosteal perilymphatic membrane. With the endosteum left intact and the endolymphatic labyrinth unexposed, the inflamed tympanomeatal membrane did not communicate directly with the perilymphatic space. As a result of such treatment of the newly created fenestra, the severity of t postoperative labyrinthitis and the frequency damage of the cochlear nerve were considerab reduced. The improvement of hearing in the cases reached as high a level as in the other cas in which the endosteum was removed. Howeve it was unfortunately observed in this series cases that both the frequency and the rapidi of osteogenetic closure of the newly create fenestra were definitely enhanced when t endosteum was left intact. The endosteum le intact acted as a support for the rapid formati of a fibrous connective tissue matrix within t bony fenestra which in turn acted as a brid for the spread of new bone formation from t freshly cut bony edges of the fenestral rim, cau ing osteogenetic closure of the gap.

2. After I had demonstrated that both t frequency and the severity of postoperative i flammation of the endolymphatic labyrinth ca be reduced by leaving the endosteum on the flo of the newly created bony fenestra intact, it o curred to me that perhaps by interposing betwe the tympanomeatal membrane and the perilymp space some tissue whose resistance to inflamma tion would be greater than that of the delica endothelial structure known as endosteum, t frequency and the severity of postoperative i flammation of the endolymphatic labyrinth cou be still further reduced and perhaps even avoide

With this objective in mind, I operated in l cases with the following technic: I created t fenestra in the bony capsule of the surgical dom of the vestibule and opened the perilymph spa by removing the endosteum. The endosteum wa removed so that it would not form a floor f the support of a fibrous connective tissue matri and thus closure of the fenestra by osteogenes was discouraged. The entire outer or perioste surface of the bony fenestra was covered wit a thinly shaved disk of cartilage prepared from piece of cartilage obtained from the anteri margin of the concha. The tympanomeatal mem brane was then placed in position to cover t cartilage-covered fenestra, in the hope that t cartilaginous disk which was interposed betwe the tympanomeatal membrane and the bon fenestra would act as a barrier to the extensio of inflammation from the tympanomeatal mem brane to the perilymph space.

A study of these cases reveals that the inci dence and the severity of postoperative inflamma tion of the endolymphatic labyrinth were bot greatly increased instead of reduced as a resu of this method of treating the newly create fenestra. The hearing improvement obtained o the operating table was not maintained thereafte

While serving the purpose of separating the tympanomeatal membrane from direct contact with the perilymph space, the cartilaginous disk in the position in which it was placed did not seal the perilymph space which was opened by removal of the endosteum. It thus permitted inflammatory exudates from the inflamed tympanomeatal membrane to reach the perilymph space and involve the endolymphatic labyrinth.

I soon realized that if cartilage or any other substance is to be interposed between the tympanomeatal membrane and the perilymph space it would have to be placed into the gap of the fenestra so that it would be in direct contact with the perilymph space instead of on top of the fenestra as a cover in contact with the outer periosteal surface of the bony vestibular capsule, in order to act as a barrier to the extension of inflammation from the tympanomeatal membrane into the perilymph space. Believing, as everybody else did, that in order to maintain continuously the hearing improvement obtained by creating a new vestibular fenestra the newly created fenestra must be kept permanently open, the thought of obliterating the gap with a substance possessing the solidity and consistency of cartilage in order to prevent the extension of inflammation from the tympanomeatal membrane to the endolymphatic labyrinth and expecting at the same time to continue to maintain the hearing improvement even temporarily seemed to be illogical and contrary to the accepted theories and principles on which the surgical treatment for the improvement of hearing was based and being developed.

The further I carried my research on the end results of fenestration operations the more obvious it became that the blame for the most untoward results could be placed directly on the belief that in order to maintain the hearing improvement it is essential to keep the newly created fenestra permanently open. By the same token of reasoning it became apparent that just as long as the newly created fenestra had to be kept open in order to improve hearing, postoperative inflammation of the endolymphatic labyrinth with secondary damage to the organ of Corti resulting from extension of inflammation from the tympanomeatal membrane could not be altogether avoided.

CAN HEARING IMPROVEMENT OBTAINED BY FENESTRATION BE CONTINUOUSLY MAINTAINED BY NOT KEEPING THE FENESTRA OPEN?

The physiologic basis for fenestration of the vestibular labyrinth in clinical otosclerosis is most likely the free mobilization of the perilymph and the endolymph whose mobility for air-borne sound has been impeded by the osseous fixation of the stapedial footplate within the oval window. Ever since it was first observed, about thirty years ago, that fistulization of the external semicircular canal in otosclerosis resulted in an immediate improvement of hearing and that this improvement disappeared as soon as the fistula closed as a result of regeneration of bone, the otologists interested in this work have striven to find means and ways of keeping the newly created fenestra permanently open, because they believed that unless it could be kept permanently open the perilymph could not be mobilized by air-borne sound and the improvement of hearing could not be maintained. Imbued with this belief and thought, they never examined the possibilities of rendering the immobilized perilymph and endolymph mobile for air-borne sound once more in a manner other than that of keeping the newly created window permanently open, despite the fact that they recognized some of the disadvantages and dangers to the immediate and continued well-being of the endolymphatic labyrinth of pursuing a policy of keeping the fenestra open.

While constantly striving to find means and ways for keeping the newly created window permanently open in order to maintain continuously the improved hearing, all failed to take cognizance of the fact that one normally hears in the presence of an oval window which is not really an open window. Nature's normal oval window is not an open window in the sense that the round window is, nor is the normal oval window open like the new oval window which otologic surgeons were for many years trying to construct. The normal oval window is not open like the fenestra nov-ovalis which they have tried to create, nor is it closed like the osteogenetically closed newly created window which they have tried to avoid. The normal oval window is closed by bone, but this bony closure is mobile, unlike the bony closure of the osteogenetically closed new oval window, which is rigidly ankylosed and immobilizable.

Anatomically, then, the normally functioning oval window is a mobile closed window. Its gap is sealed by the bony base of the stapes, which fits the bony margins of the fenestra snugly, separated from them only by the annular ligament which frames and holds the entire circumference of the bony base of the stapes. The inner surface of this bony footplate which faces the vestibular perilymph space is cartilaginous.

Thus it can be readily seen that the normal oval window is not an open but a closed window

and that the closure, though bony, is nevertheless mobile. As long as the oval window retains this state of anatomic construction, wherein the bony footplate which closes the gap of this window remains mobilizable by air-borne sound, hearing for such sound remains normal. However, when in otosclerosis this footplate becomes fixed and rigid as a result of proliferation of the bony tumor beyond the endosteal or the periosteal layer of the bony wall of the oval window, immobility for air-borne sound ensues and hearing becomes impaired.

Therefore, it can readily be understood that hearing becomes impaired in otosclerosis not because the window is no longer open, for the normal oval window is never open, but rather because the structure normally closing the oval window becomes rigidly fixed and ceases to be mobile.

Along these lines of reasoning it appeared to me that to maintain the improved hearing following the creation of a new fenestra it is perhaps not necessary to keep the fenestra open and that one may close the gap and still maintain the improved hearing, provided one can keep the closure mobile.

Considering the anatomic construction of the oval window and comparing the normal functional response of the mobile bony stapedial footplate under the impact of air-borne sound with the impeded response of the stapedial footplate which has been ankylosed by an otosclerotic process, I could not help but feel that perhaps a newly created fenestra which closes as a result of regeneration of bone becomes functionally impeded not because bony closure of the gap took place but because the bony growth which closed the gap is ankylosed to the rim of the fenestra and remains immobile. Perhaps if this new growth were mobile instead of ankylosed, the improvement of hearing obtained by fenestration would not recede.

IS IT BONY CLOSURE OR IS IT ANKYLOSED BONY CLOSURE WHICH PREVENTS THE MAINTENANCE OF THE HEARING IMPROVEMENT?

Because of the generally unsatisfactory end results obtained by removing the newly regenerated bone from an osteogenetically closed newly created fenestra, I decided that in revising several osteogenetically closed fenestras I would free the newly formed bony mass from its bony attachment to the walls of the labyrinthine fenestra and after having rendered it mobilizable inward toward the perilymph space I would leave it in position within the fenestra instead of removing it as heretofore practiced.

I hoped as a result of such treatment of the osteogenetically closed new fenestra to learn:

1. Whether by mobilizing the rigid, immobi… newly formed bony mass which obliterates t… fenestral gap and leaving it in situ the hearin… improvement which was lost as a result of t… postoperative formation of this rigid bony ma… could be restored. This would tell me wheth… the bony mass proper offers impedance to t… mobilization of the perilymph and the endolym… for the transmission of air-borne sound … whether the impedance results only because t… newly regenerated bone within the gap is ank… losed to the bony rim of the fenestra. Such i… formation would help to determine once and f… all whether in order to maintain the improve… hearing the newly created fenestra must be ke… open or may be closed and obliterated by a sto… ple as soon as it is opened, provided the stopp… used can remain mobile.

2. Whether the mobilized bony mass left … situ within the fenestra, thus separating the pos… operatively inflamed tympanomeatal membra… from direct contact with the perilymph spac… can greatly reduce or even prevent postoperati… inflammation of the endolymphatic labyrin… with consequent damage of the cochlear nerv… which only too frequently resulted in those cas… in which the bony mass was removed durin… revision of the fenestra. This would also infor… me whether a stopple obliterating the fenestr… gap could accomplish what a cartilage disk us… as a lid to cover the fenestra, instead of oblit… ating the gap, failed to do.

3. Whether such a newly formed rigid bon… mass after having been rendered free and mobi… and left in situ will osteogenetically unite wi… the bony walls of the fenestra and become rig… and immobile once again.

I performed six revisions, employing the fo… lowing technic:

After separating and lifting the tympan… meatal membrane carefully, I exposed to view t… osteogenetically closed fenestra nov-ovalis wit… out opening the tympanic air space. The perio… teal or outer surface of the newly formed bon… mass which obliterated the gap of the new fe… estra was shaved with an electrically driven de… tal plug finishing burr until it became detache… and separated from the bony rim of the former… created fenestra in the bony labyrinthine capsu… of the vestibule. By moderately increasing th… pressure of the revolving burr over the out… surface of the newly formed bony mass, whi… is readily distinguishable from the normal bon… labyrinthine capsule, the entire mass was di… placed inward toward the perilymph space b… yond the inner or endosteal surface of the bon… rim of the fenestra. The thus mobilized mass … newly regenerated bone was left in situ with…

the gap, and the tympanomeatal membrane was replaced in position to cover the fenestra with the mobilized regenerated bone in position within the gap. The following observations were made in the 6 cases in which the fenestra was thus revised:

1. Hearing was improved as soon as the newly formed bony mass was freed from its attachments to the rim of the fenestra in the bony labyrinthine capsule. The improvement of hearing obtained by testing at the operating table was as good with the mobilized bony mass in situ as it was when the fenestra was first created during the original operation, with the gap left widely open.

2. Hearing remained improved after the fenestra had been covered with the tympanomeatal membrane despite the fact that the mobilized newly formed bony mass was left in situ within the gap, interposed between the tympanomeatal membrane and the perilymph space.

3. Hearing continued to remain improved in these cases for periods ranging from two to three months postoperatively; then the newly formed bony mass became ankylosed and immobilized once more as a result of new bone regeneration and the hearing again receded to the preoperative level. However, the fact that hearing remained improved as long as the bony mass within the fenestra remained mobile served as proof conclusive that a newly created fenestra in the vestibular labyrinth need not be kept open to improve hearing in clinical otosclerosis and that hearing can be improved by closing the fenestra if a method of closing can be devised whereby the closure is and remains mobile.

4. The newly regenerated bone which was mobilized and left in situ within the fenestra to prevent direct contact and communication between the tympanomeatal membrane and the perilymph space definitely became a barrier to the spread of inflammation from the tympanomeatal membrane to the endolymphatic labyrinth. Postoperative labyrinthitis with secondary damage to the organ of Corti, which was common after revisions when the newly formed bony mass was removed, did not occur in any of the cases in which the newly formed bony mass was mobilized and left in situ.

WILL A NEWLY CREATED FENESTRA IN WHICH THE GAP HAS BEEN IMMEDIATELY OBLITERATED BY A MOBILE STOPPLE DURING THE ORIGINAL OPERATION BEHAVE THE SAME AS A FENESTRA IN WHICH THE NEW BONE FORMATION HAS BEEN MOBILIZED BY A REVISION?

Having proved that it is not necessary to keep newly created fenestra open in order to maintain the improved hearing obtained by fenestration of the vestibular labyrinth in clinical otosclerosis and having also shown conclusively that the interposition of a bony stopple in the gap between the perilymph space and the tympanomeatal membrane does prevent severe postoperative inflammation of the endolymphatic labyrinth, it soon became obvious that if a stopple possessing properties conducive to its remaining mobile within the fenestra were to be employed for obliterating the gap instead of the fractured inward lid of newly formed bone, which invariably became ankylosed and immobile, a new and great advance in the surgical treatment for otosclerosis would have been reached.

The substance chosen to be used as a stopple will have to be resistant to inflammation extending to it from the tympanomeatal membrane and inert and nonirritating to the endolymphatic labyrinth, with both of which it will come in contact. Bone used as a stopple will always act as a graft and stimulate regeneration of bone from the bony walls of the fenestra unless it is framed within a structure like the annular ligament which frames the bony rim of the normal oval window and thus keeps it constantly mobile. Since the surgical creation of a bony stopple framed in such a ligament is still beyond human endeavor and reach, the thought of employing bone as a stopple had to be dismissed.

Impressed with the fact that nature chose cartilage, and apparently for a good reason, to line the inner surface of the footplate of the stapes, the surface which faces the perilymph, I decided to use cartilage again, but this time not in the form of a lid to cover the periosteal surface in the manner in which I formerly unsuccessfully employed it in an attempt to prevent postoperative inflammation and damage to the organ of Corti, but in the form of a stopple to be inserted into and to obliterate the gap of the newly created fenestra.

I performed the fenestra nov-ovalis operation in a series of cases as follows:

After fenestrating the bony capsule of the surgical dome of the vestibule I removed the presenting surface of endosteum and exposed to view the endolymphatic labyrinth within the perilymph space. After measuring the length and the width of the fenestral gap I resected a small piece of flat cartilage from the anterior margin of the concha forming the outer boundary of the membranous portion of the posterior wall of the external auditory canal. The edges of the resected piece of cartilage were then shaved to the measurements of the gap so that when introduced into the fenestra it would snugly fit the contour of the bony rim of the

fenestra. The cartilaginous lid was then placed into the fenestra so that it completely obliterated and closed the newly created window in the bony capsule of the surgical dome of the vestibule. The cartilage-closed fenestra was then covered with the tympanomeatal membrane.

The following were the observations made in the cases in which the operation was thus performed:

1. Closure of the gap of the newly created fenestra with a cartilage stopple did not interfere with the hearing improvement obtained immediately after fenestration of the bony capsule of the vestibular dome. The final degree of the improvement obtained and maintained in these cases was as high as the best hearing improvement obtained when the fenestra was left open.

2. In none of these cases did postoperative inflammation of the endolymphatic labyrinth occur, and postoperative damage of the organ of Corti was avoided in all, as evidenced by the fact that hearing never receded even temporarily to a level lower than it was preoperatively. The experiment convinced me that by obliterating the gap of a newly created fenestra with a cartilaginous stopple the otologic surgeon can prevent one of the major causes of defeat, namely, damage to the organ of Corti secondary to postoperative inflammation of the endolymphatic labyrinth, which could not be prevented by the open fenestra treatment or even by employing cartilage as a cover for the fenestra.

3. In 8 of the 10 patients thus treated the hearing was continuously maintained at the highest originally obtained level of improved hearing.

The fact that 8 of 10 patients continued uninterruptedly to retain their improved hearing despite the fact that the fenestra was not kept open convinced me that to maintain the improvement in hearing it is not essential to keep the fenestra open and that it may be closed again without ever interfering with the improvement of hearing obtained, provided the stopple employed to close it remains mobile.

4. In 2 patients the response to the fistula test vanished and was not obtainable at about the end of three months, suggesting that the cartilaginous stopple was no longer mobile. The hearing in these 2 patients receded to the preoperative level.

Osteogenesis immobilized the bony stopple in every one of the 6 cases in which the mobilized bony lid was employed to close the gap, whereas with employment of cartilage as a stopple only in 2 of 10 cases was the stopple rendered immobile.

Though already convinced as a result of t last investigation that closing the fenestra stead of leaving it open was advantageous a that the advantages accruing to the patient a result of the closed fenestra treatment w already sufficient to warrant the abandonment the open and the adoption of the closed fenes method, I nevertheless decided to carry my search still further before adopting this meth of fenestration to the exclusion of all other me ods I had hitherto employed. This decision press my investigations still further was ba on a feeling that the principle of employing stopple for the obliteration of the gap of a new created fenestra somehow and somewhere offer an unusual opportunity, the type of which w never offered by the open fenestra treatment i the deliberate surgicophysical consistent preve tion of osteogenetic closure of every new created fenestra, which I have been investigati for many years.

WHY AND HOW DID THE ORIGINALLY MOBI CARTILIGE STOPPLE BECOME IMMOBILE IN TWO OF THE TEN EARS THUS TREATED?

I investigated the 2 cases in which the heari receded to the preoperative level and in whi clinical evidence of immobility of the cartila stopple was obtainable. The findings were t same in both cases. Inspection of the fenes revealed a growth of newly formed bone bridgi the outer or periosteal surface of the bony r of the fenestra. After this outer thin layer bone was removed with the electrically driv dental plug finishing burr, the cartilagino stopple was exposed to view and observed to still in position within the gap. Hearing i provement immediately followed the removal the periosteal layer of new bone. The cartila stopple showed no evidence of having partic pated in the osteogenetic repair. It was not a tached to the new periosteal bony lid nor to t bony rim of the fenestra and was perfectly mob in situ. No new bone was observed either in t bony rim of the fenestra or on the inner or endo teal surface of the fenestra. Whereas the out or mastoid aspect of the cartilage stopple w observed to occupy a position level with that the periosteal or outer surface of the bony ri of the fenestra, the inner or perilymphatic aspe of the stopple was found to be extending in the perilymph space approximately 0.5 to 1 m beyond the endosteal or inner surface of the bo rim of the fenestra. When the fact that rege eration of bone in both of these cases took pla only in the periosteal or outer mastoid aspe

of the gap was correlated with the relation of the position of the cartilage stopple within the gap to the periosteal and endosteal surfaces of the bony rim of the fenestra, I could not help but suspect that the fact of the new bone formation having taken place in both cases only on the periosteal surface of the fenestra was not just a coincidence and that perhaps the difference in the position of the cartilage cork in its relation to the periosteal and endosteal surfaces of the bony rim of the fenestra may have been the cause for osteogenesis taking place only in the outer or periosteal surface.

I therefore reasoned that osteogenesis did not take place within the fenestral gap proper or bridge the endosteal or perilymphatic surface of the fenestra because the cartilage stopple in these 2 cases obliterated the gap and extended beyond the endosteal surface of the bony rim of the fenestra into the perilymph space, thus preventing the formation of a fibrous tissue matrix either in the gap proper or on the endosteal surface of the fenestra, for the spread of osteogenesis from the freshly cut bony walls.

By the same reasoning it seemed plausible to assume that since the outer or mastoid aspect of the cartilage stopple in these 2 cases occupied a position within the gap on a level with the outer or periosteal freshly cut surface of the bony rim of the fenestra a fibrous connective tissue matrix did form extending from the periosteal or outer margin of the bony rim of the fenestra to bridge the outer aspect of the gap, thus permitting osteogenesis from the periosteal layer to take root and spread across the outer aspect of the gap and thus prevent the mobilization by airborne sound of the cartilage stopple which remained mobile within the gap.

If the foregoing hypothesis is true, then if a cartilage stopple when employed to obliterate the gap of the newly created fenestra is introduced in such a manner that both its inner surface, facing the perilymph space, and its outer surface, facing the epitympanum, would respectively reach and extend approximately 0.5 mm. beyond the endosteal and periosteal surfaces of the bony rim of the fenestra, it should prevent the formation of a fibrous connective tissue matrix in the gap and on the endosteal and periosteal surfaces of the bony rim of the fenestra as well. Osteogenesis within the freshly cut bony walls of the fenestra should thus be physically prevented from taking root and spreading with consequent obliteration of the gap, and the bridging by new bone of the outer and inner surfaces of the bony rim of the fenestra should be avoided.

HOW CAN IMMOBILE OSTEOGENETIC CLOSURE OF A NEWLY CREATED VESTIBULAR FENESTRA BE DELIBERATELY AND CONSISTENTLY PREVENTED?

Ankylosed closure by newly formed bone of a newly created fenestra in the bony vestibular capsule, which renders the perilymph immobile after it has been mobilized by fenestration, can be deliberately and consistently prevented and permanent mobility of the perilymph can be obtained at will not by insistently keeping the fenestra open but by immediately corking and permanently closing the newly created fenestra with a mobile stopple. Such a stopple must be created from a substance which is inert and will not irritate the endolymphatic labyrinth with which it may come in contact, must possess a consistency sufficient to impede physically osteogenesis within the circumference of the freshly cut bony rim of the fenestra, must be smooth and light in weight so that it will be easily mobilizable in situ and must be introduced and held in the fenestra in a manner prescribed which will serve and tend to prevent the formation of a fibrous connective tissue matrix for the spread of new bone from any of the histologic layers of the freshly cut bony walls of the fenestra. Auricular cartilage, which is a solid, tough, resistant, smooth, light and elastic substance, is best suited for employment as such a mobile stopple.

I sporadically operated in cases here and there during the last two and a half years at various intervals, employing the following new technic and closely studying and observing the end results in each case for periods of varying length before attempting to repeat the procedure, until 50 cases were completed and studied.

TECHNIC OF THE LEMPERT FENESTRA NOV-OVALIS WITH MOBILE STOPPLE

The fenestra nov-ovalis is created in the surgical dome of the vestibule with the technic which I have described.[6] The spina helicis of the auricular cartilage is exposed to view and carefully resected with a fine sharp scalpel. With the aid of Zeiss magnifying glasses, the cartilaginous spina helicis is then carefully trimmed and shaved until it is about 2 mm. thick and conforms in width and length to the fenestra nov-ovalis. The dimensions are determined with specially devised measuring instruments. This specially prepared solid, smooth and elastic cartilaginous obturator stopple is then placed over the fenestral gap, and

6. Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880-912 (Nov.) 1941.

with a specially devised instrument it is squeezed into the gap between the freshly cut margins of the bony rim of the fenestra, like a cork into the opening of a bottle. The inner surface of the cartilaginous stopple should extend approximately 0.5 mm. beyond the inner or endosteal surface of the bony rim of the fenestra into the perilymph space of the vestibule, while the outer surface of the stopple should protrude about 0.5 mm. outward beyond the periosteal surface of the bony rim of the fenestra into the epitympanum. The cartilage-stoppled fenestra is covered with the tympanomeatal membrane, which is then held in position by a paraffin-impregnated mesh gauze anchor and paraffin.

The patients operated on with this new technic were carefully chosen. Only those with overabundant evidence of a reservoir of residual untapped cochlear nerve function large enough to permit the restoration of practical, serviceable hearing were chosen. This special precaution was taken so that the end result obtained, whether good or bad, could be evaluated properly and accredited solely to the technic employed. In case of failure the technic could be held as the sole factor responsible for the failure.

The following observations were made in the cases in which fenestration was done with this new technic:

1. Postoperative serous labyrinthitis with secondary damage to the organ of Corti was prevented in every case, thus doing away with the menace of early postoperative defeat of one's efforts for maintaining continuously the improvement of hearing which followed the creation of the new fenestra. By immediately corking the fenestra with the cartilage stopple one sealed the perilymph space and separated it from direct contact with the tympanomeatal flap. Thus postoperative inflammation of the flap was prevented from extending to the endolymphatic labyrinth within the perilymph space. Auricular cartilage, being inert, when coming in contact with the endolymphatic labyrinth behaves like the cartilage-covered inner surface of the stapedial footplate. It is histologically most suitable to the continued well-being of the endolymphatic labyrinth.

2. Hearing was improved to the 30 decibel level or higher in every case and continuously maintained at that level despite the fact that the fenestra was closed instead of kept open. It is now more than two years since the first patient was operated on by this new method.

The drop in the hearing for high frequencies which often followed fenestration when the fenestra was kept open did not take place in any of these cases in which the fenestra was immediately and permanently closed with a mobile cartilage stopple.

The mobile cartilage stopple simulates functionally the normal stapedial footplate. It dampens loud air-conducted sound and protects the organ of Corti from acoustic trauma.

3. To date there are no signs or symptoms of osteogenesis in the newly created fenestra in any of these cases. The cartilage stopple has remained in situ and continues to be mobile. The lightest contact with the area of the tympanomeatal flap which covers the stopple elicits nystagmus. The improved hearing continues at its high level, proving that to maintain improved hearing following fenestration it is not essential that the newly created fenestra remain open and that it is better to close it, provided such closure, like that of the normal stapedial footplate, remains mobile.

By obliterating the fenestral gap with a cartilage stopple and permitting this stopple to extend beyond the endosteal and periosteal layers of the bony rim of the fenestra the formation of a fibrous connective tissue matrix for both the start and the spread of osteogenesis from the bony rim of the fenestra was made histologically and physically impossible. It is therefore logical to believe that rigid and immobile osteogenetic closure of the newly created fenestra was thus deliberately and consistently avoided in these cases. It is not unlikely, and further investigation may prove, that a fibrous structure simulating in function the annular ligament is eventually formed between the cartilage stopple and the bony rim of the fenestra.

Because of my experience to date with this new technic and the logic by which this technic was originally conceived, I strongly believe that this new method of fenestrating the labyrinth for the cure of clinical otosclerosis is the answer to the two major causes of defeat encountered in pursuing the tradition of trying to keep the newly created fenestra open. Confident that by immediately closing the newly created fenestra with a mobile cartilaginous stopple damage to the organ of Corti as a result of postoperative serous labyrinthitis and rigid immobile osteogenetic closure of the fenestra can be both deliberately and consistently prevented, I am now adopting and employing this closed method of fenestration exclusively with the knowledge and understanding that only time and a large series of cases will eventually serve to prove my contention that the best way to keep a fenestra nov-ovalis permanently mobile is not by attempting to keep it open

Fig. 5.—

Fig. 5.—*A*, normal auricle. The antauricular suprameatal triangle is seen. *B*, first antauricular incision.

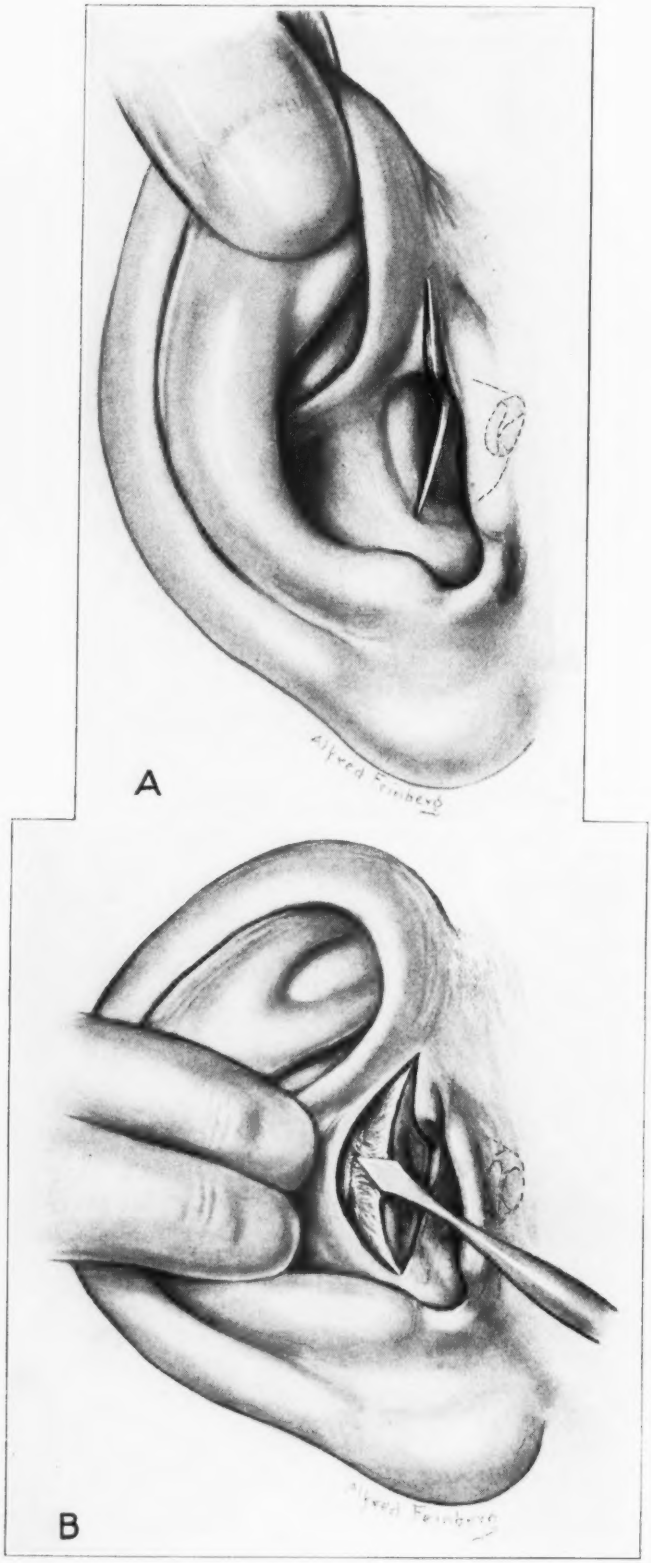

Fig. 6.—*A*, second antauricular incision. *B*, third antauricular incision.

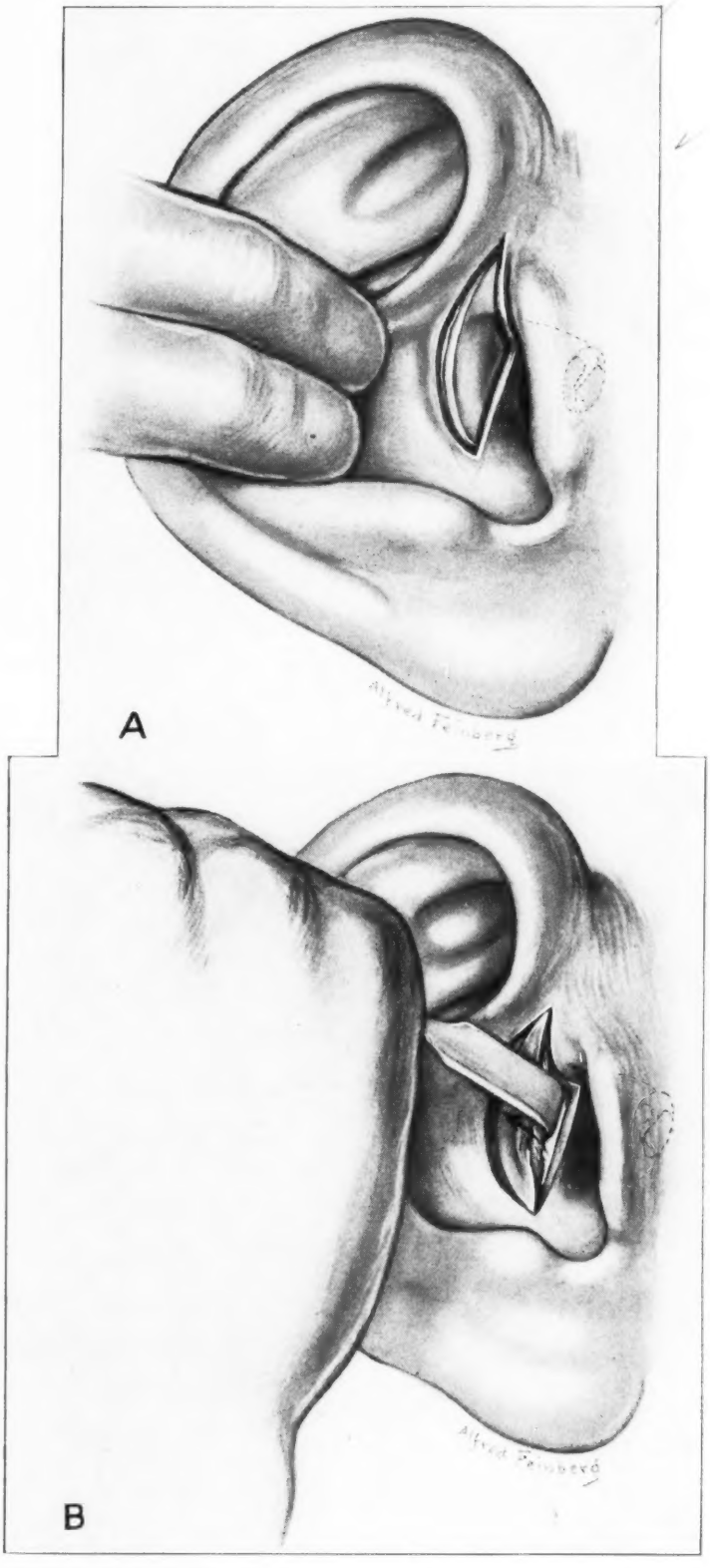

Fig. 7.—*A*, all three incisions. *B*, subperiosteal elevation of a triangular cutaneous flap.

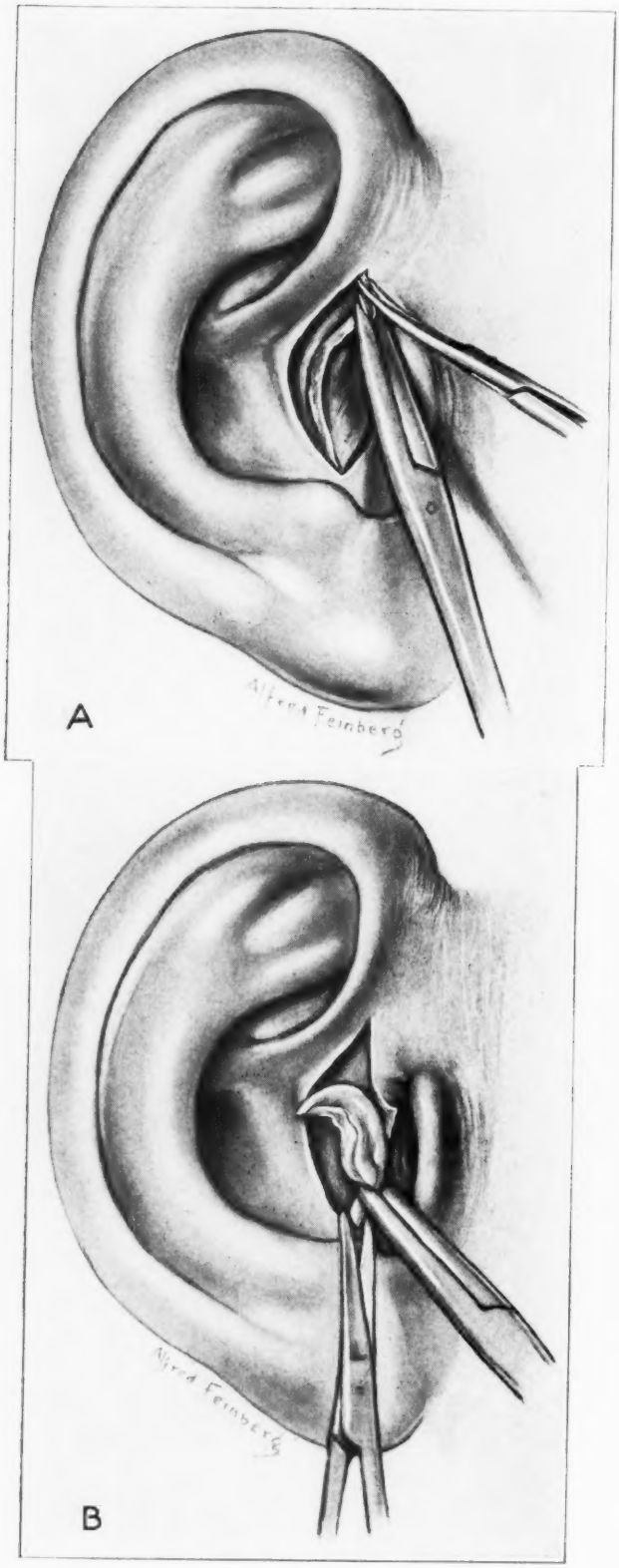

Fig. 8.—Excision of the elevated flap: *A*, step 1. *B*, step 2.

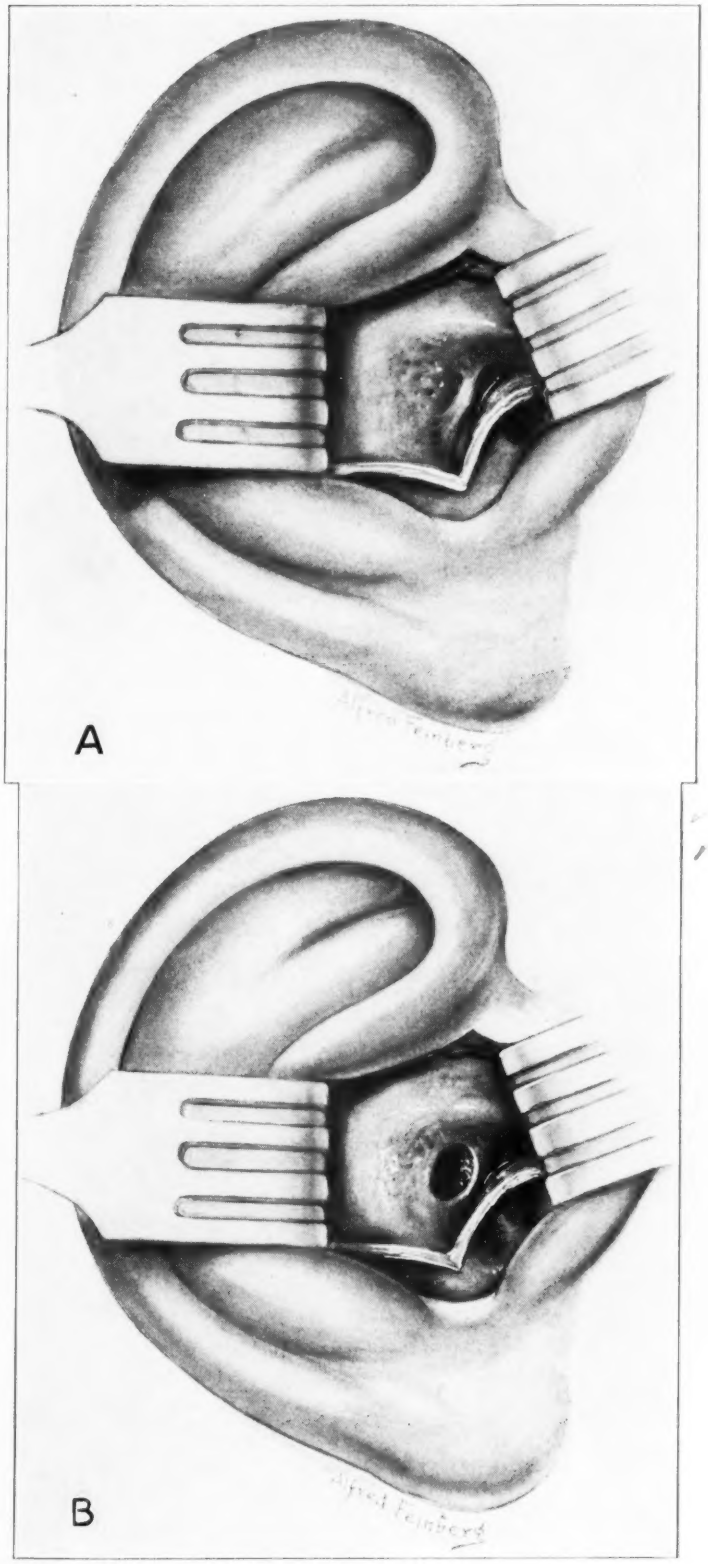

Fig. 9.—*A*, antauricular exposure of the mastoid cortex. The spina Henle, the fossa mastoidea and the cutaneous lining of the posterior and superoposterior bony walls of the external auditory canal are seen. *B*, opening into the mastoid antrum.

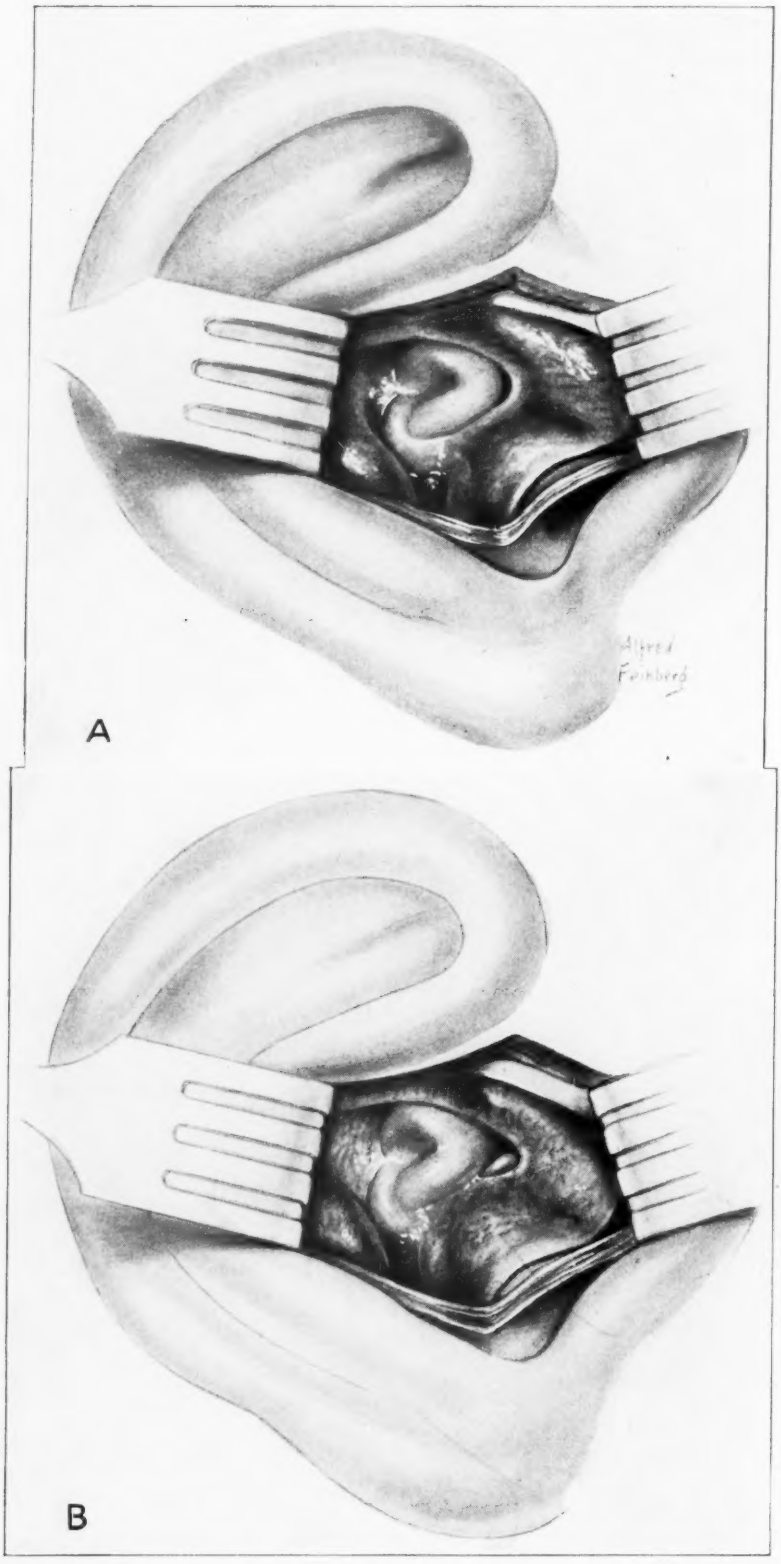

Fig. 10.—Exenterated mastoid cavity. The lateral, superior and posterior semicircular canals are sharply defined. *B*, short process of the incus exposed to view.

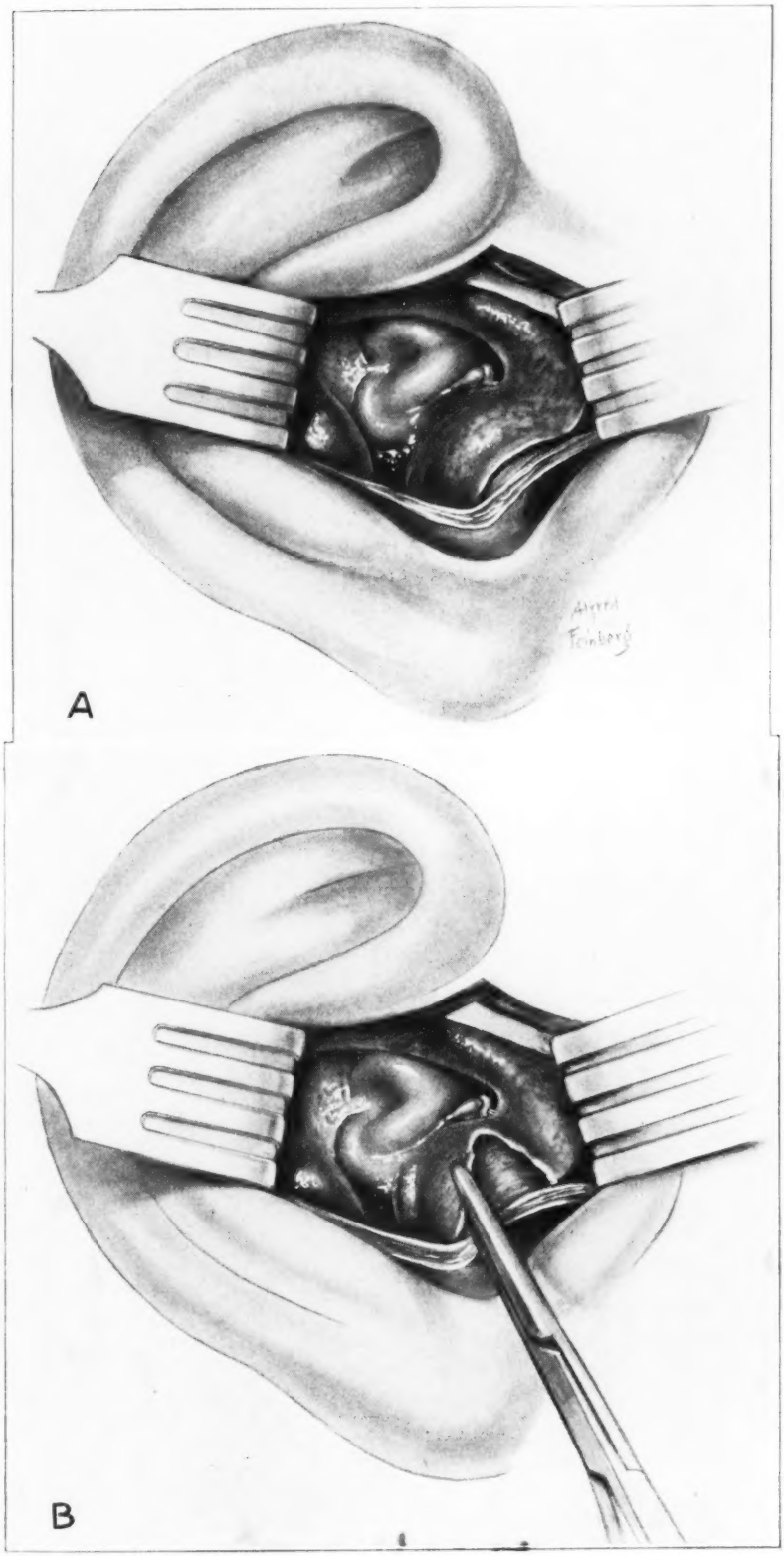

Fig. 11.—*A*, incudomalleolar joint exposed to view. *B*, removal of posterior and superoposterior bony walls of external auditory canal—step 1.

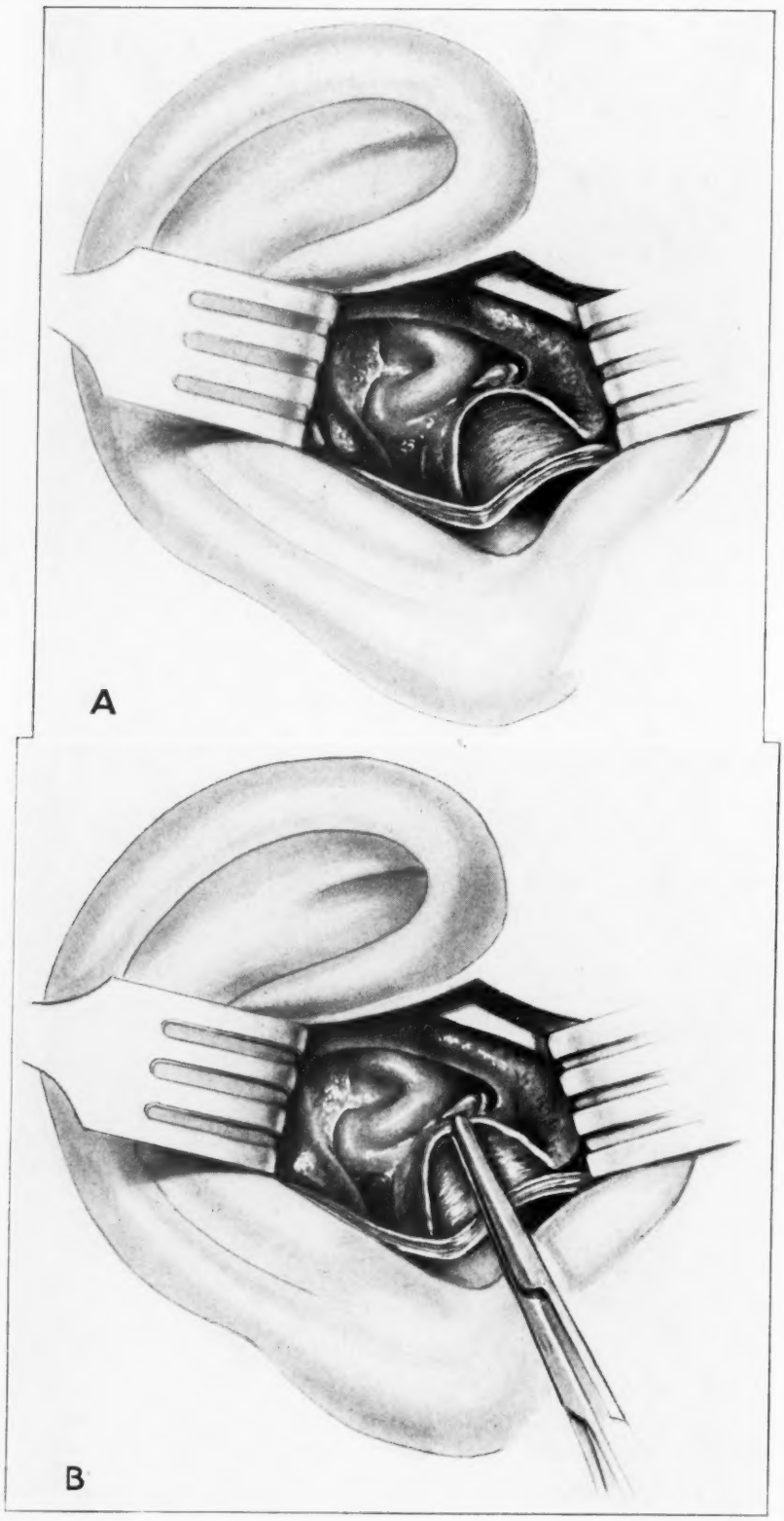

Fig. 12.—*A*, removal of posterior and superoposterior bony walls of the external auditory canal—step 2: bony canal walls are removed, leaving the cutaneous lining intact. *B*, removal of the bony bridge lateral t ossicles to expose the notch of Rivinus.

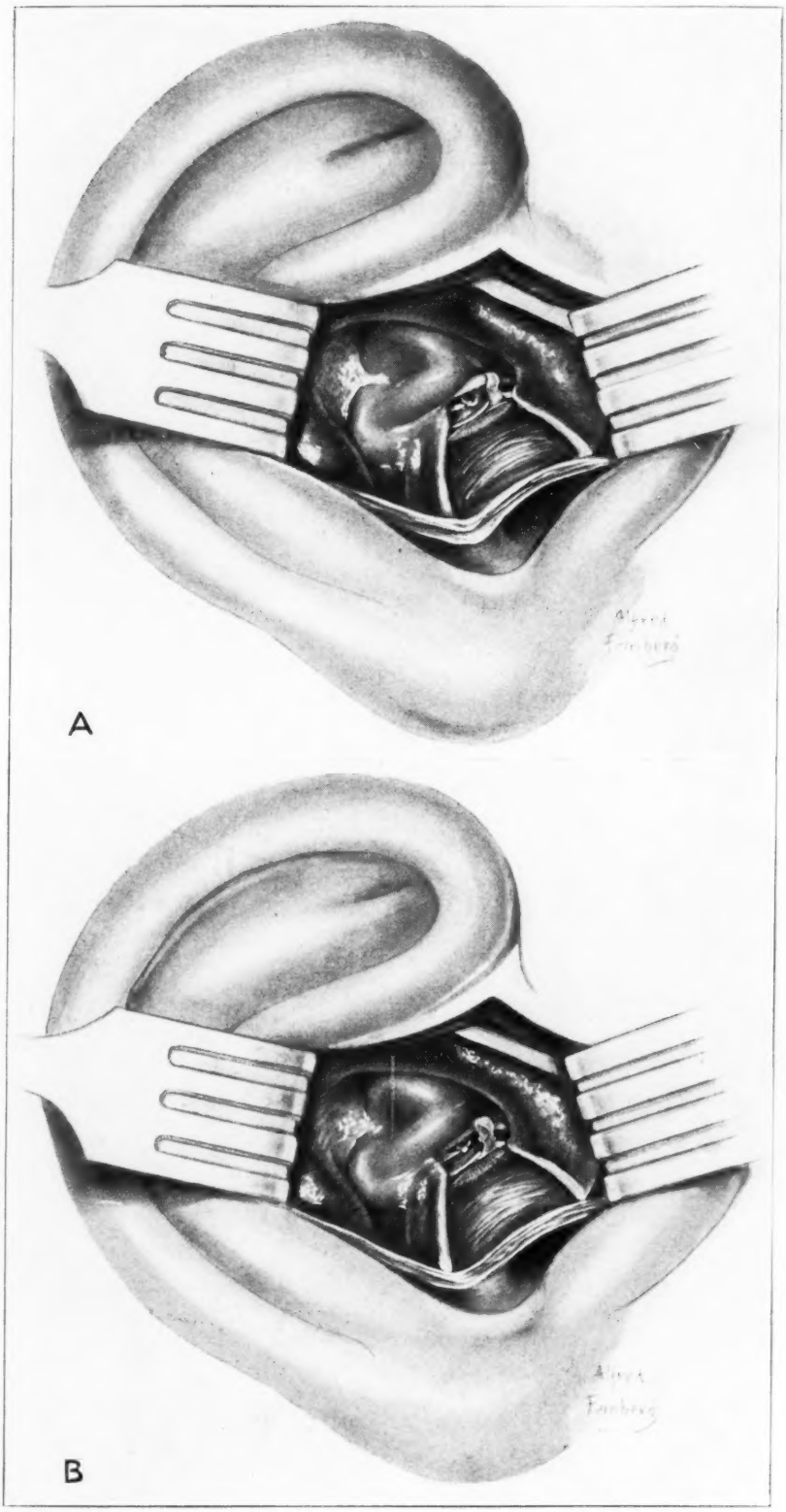

Fig. 13.—*A*, notch of Rivinus widened, exposing to view the incudomalleolar joint, the anterior malleolar ligament, the incudostapedial joint, the stapedius muscle, the oval and round windows, the cochlear promontory and the chorda tympani nerve. *B*, incus removed; malleus in position exposing to view the tympanic portion of the facial canal.

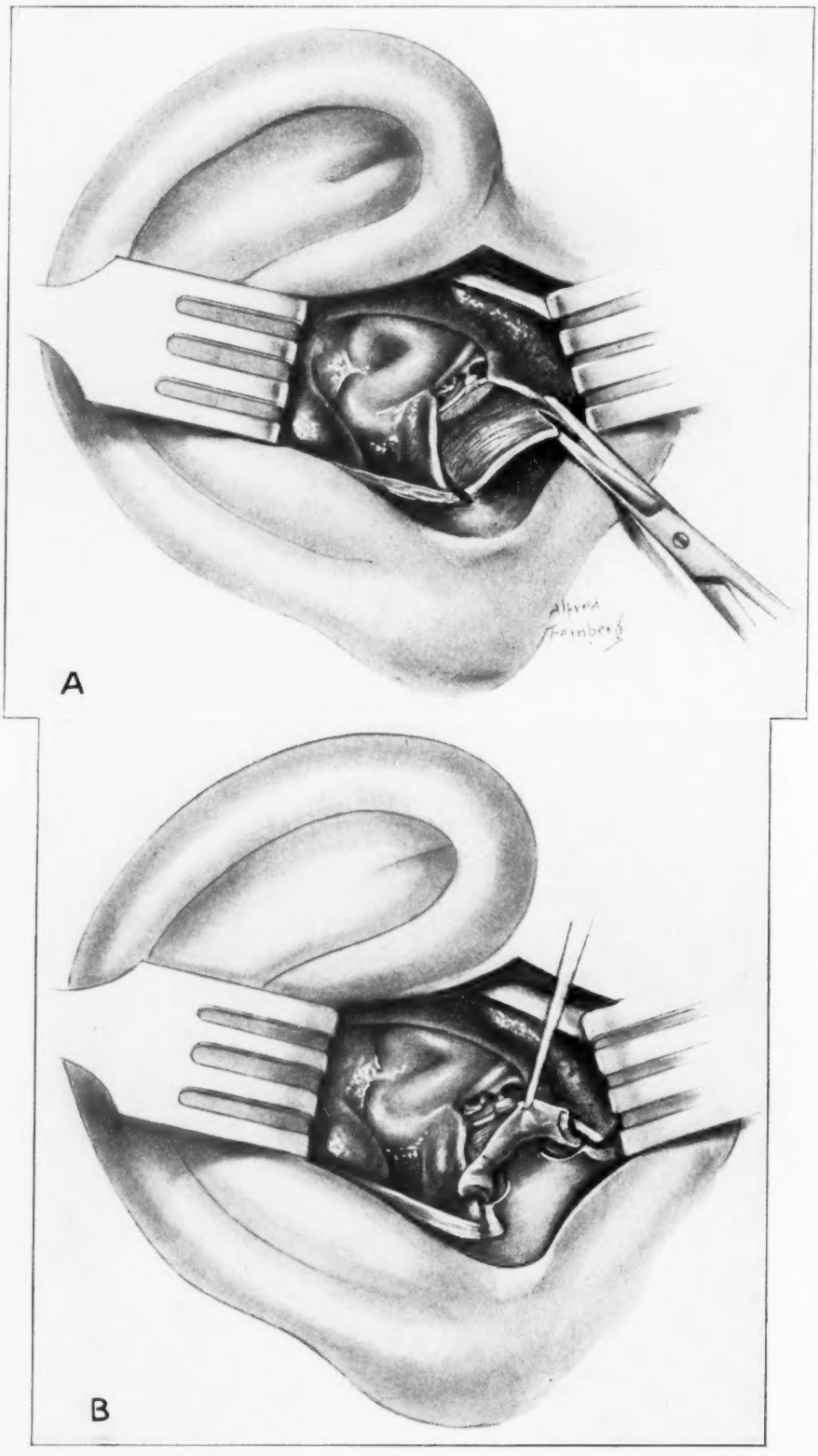

Fig. 14.—*A*, head and neck of the malleus removed, leaving the handle of the malleus in position. Exp to view are the tympanic and epitympanic portions of the facial canal and the processus cochleariformis with tendon of the tensor tympani muscle attached to the handle of the malleus. Note beginning of incisions of cutaneous lining of the canal for the creation of a tympanomeatal flap. *B*, incisions of the cutaneous li carried deeper anterosuperiorly and inferiorly into the external auditory canal to its junction with the tymp membrane.

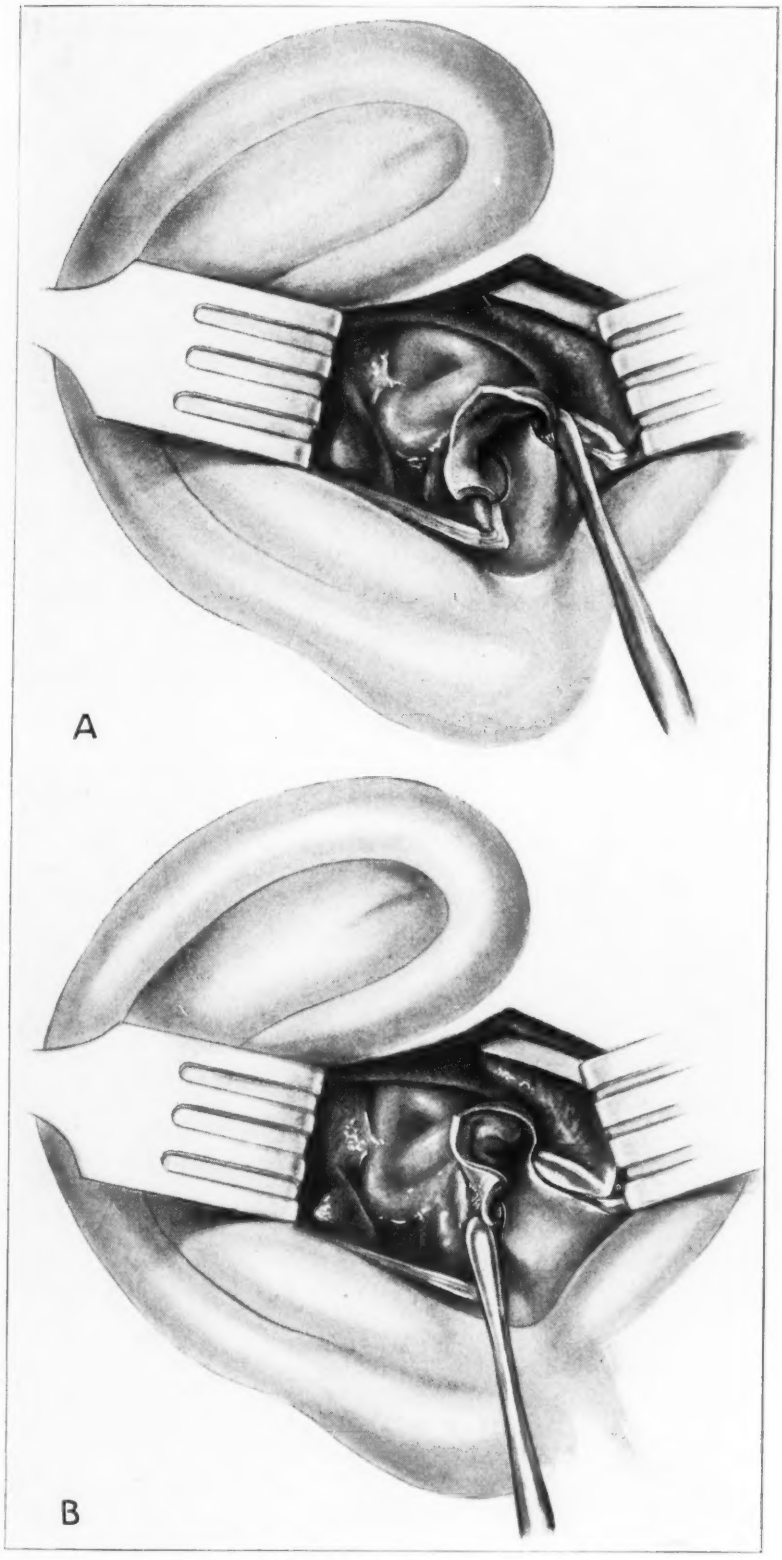

Fig. 15.—*A*, freeing of the cutaneous lining from the anterosuperior bony canal wall down to the tympanic membrane. *B*, freeing of the cutaneous lining from the inferior bony canal wall down to the tympanic membrane.

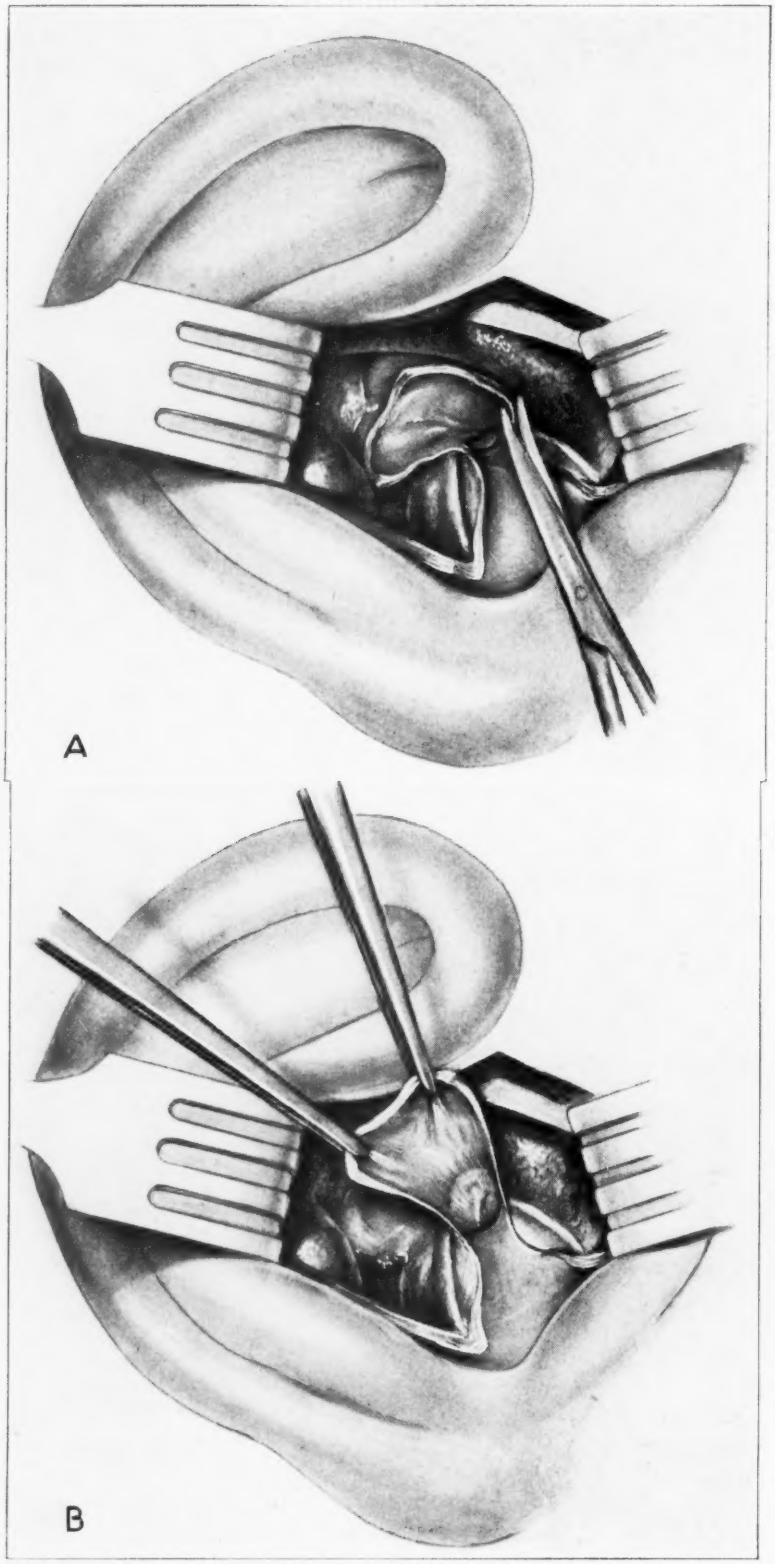

Fig. 16.—*A*, severance of restraining fibers anteriorly at the point of attachment to the tympanic membrane liberate the flap for proper adaptation over the fenestra to be created. *B*, liberated tympanomeatal flap, consis of the cutaneous lining of the external auditory canal, attached to the tympanic membrane by its dermal la

Fig. 17.—Fenestra nov-ovalis completed.

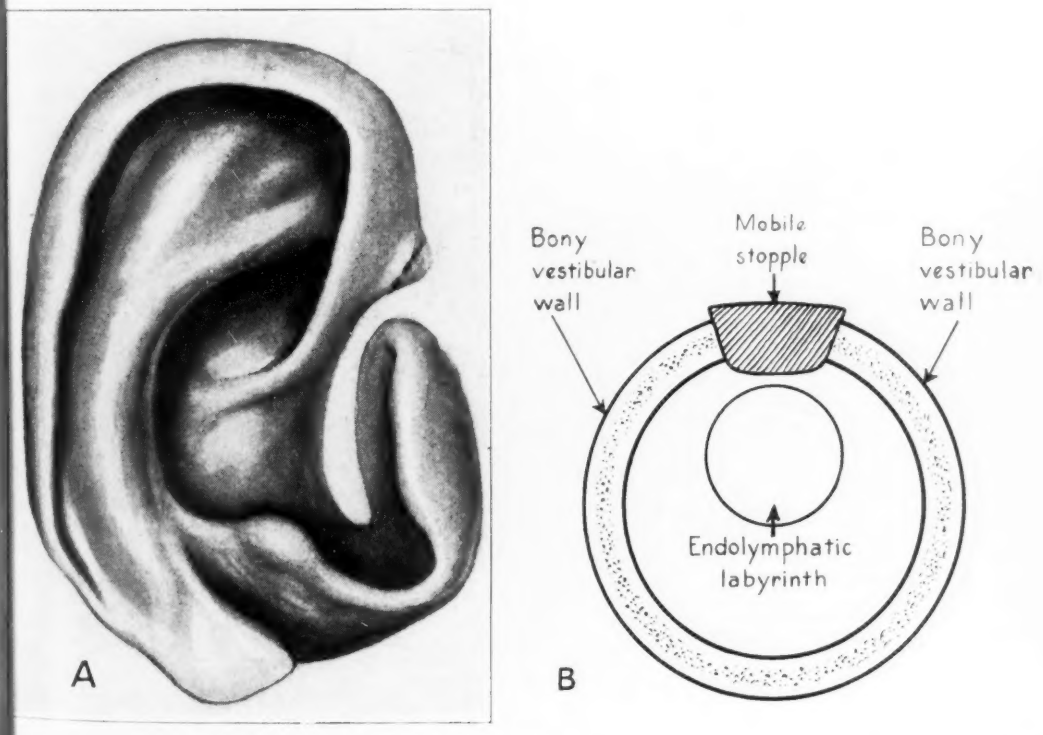

Fig. 18.—*A*, removal of the spina helicis for the creation of a stopple. *B*, schematic diagram showing the cartilaginous stopple in situ within the fenestral gap and its relationship to the bony vestibular wall, the epitympanic space, and the perilymphatic space.

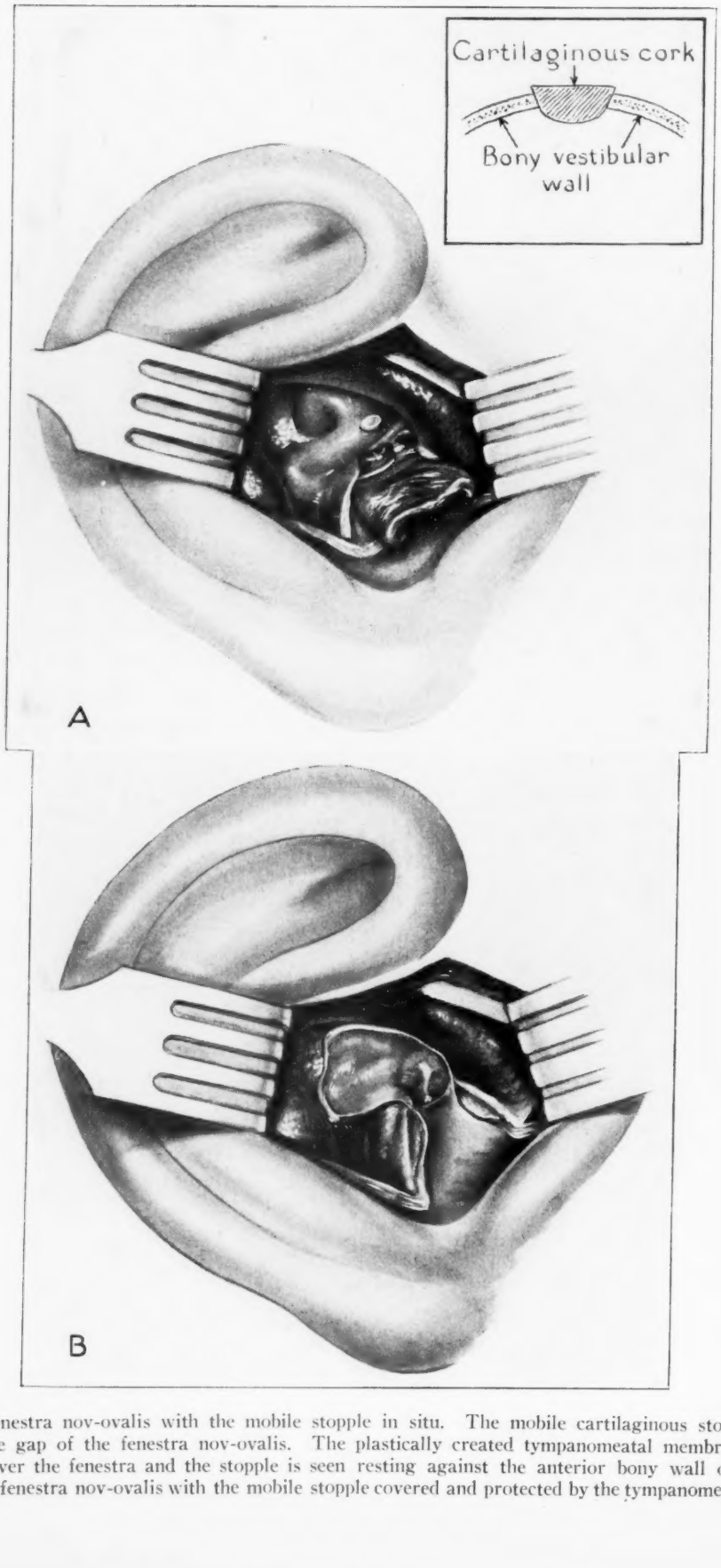

Fig. 19.—*A*, fenestra nov-ovalis with the mobile stopple in situ. The mobile cartilaginous stopple is se position within the gap of the fenestra nov-ovalis. The plastically created tympanomeatal membrane which be employed to cover the fenestra and the stopple is seen resting against the anterior bony wall of the exte auditory canal. *B*, fenestra nov-ovalis with the mobile stopple covered and protected by the tympanomeatal membr

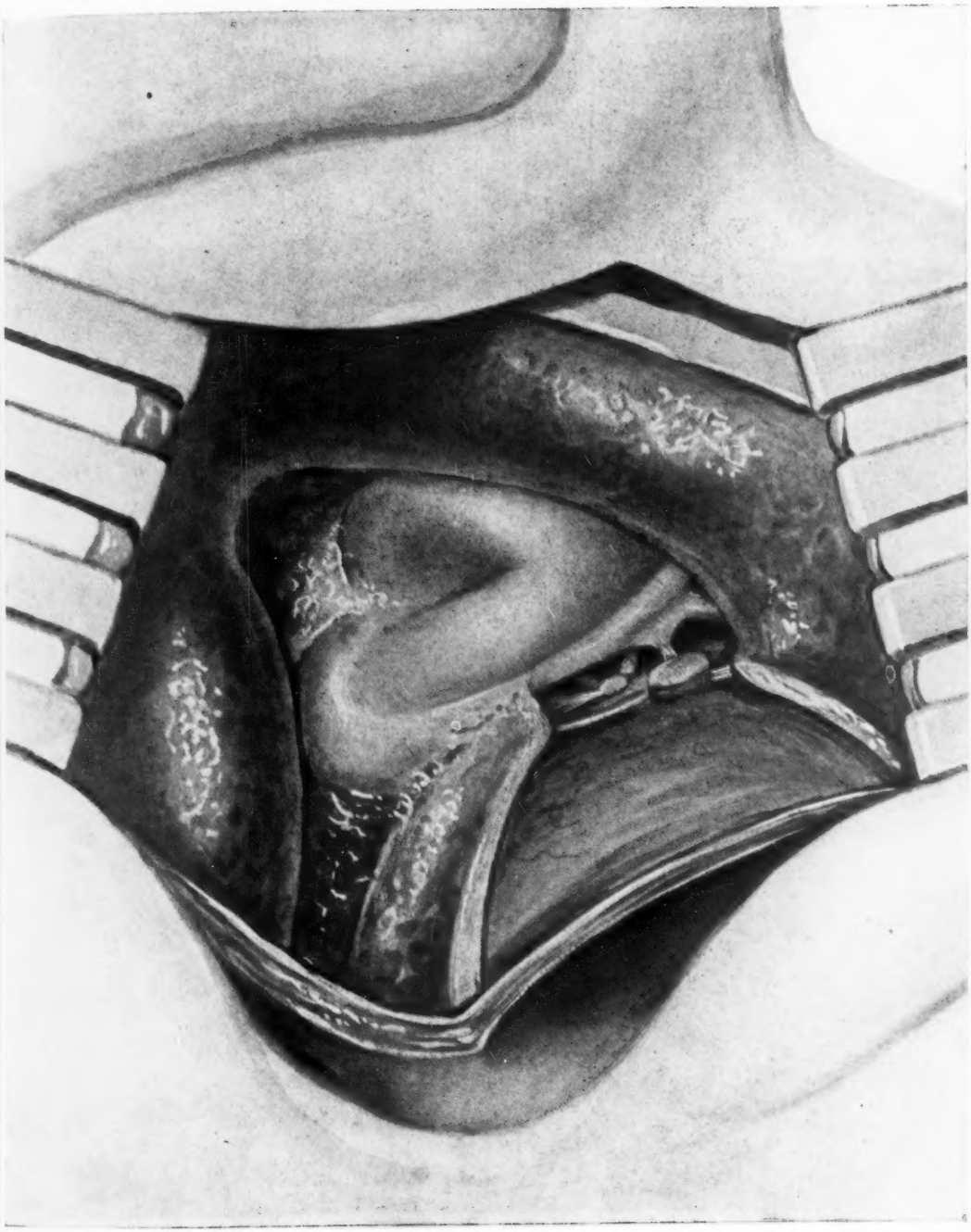

Fig. 20.—Operative field ready for the creation of the fenestra nov-ovalis. The mastoid cavity is completely exenterated. The lateral, superior and posterior semicircular canals are sharply defined. The surgical dome of the vestibule is exposed to view, and one may see the tympanic and epitympanic course of the facial canal, the processus cochleariformis with the tendon of the tensor tympanic muscle attached to the handle of the malleus, the anterior malleolar ligament and the chorda tympani nerve, the stapes in position with the stapedial tendon and the cochlear promontory.

The cutaneous lining of the external auditory canal is preserved for the plastic construction of the tympano-meatal flap.

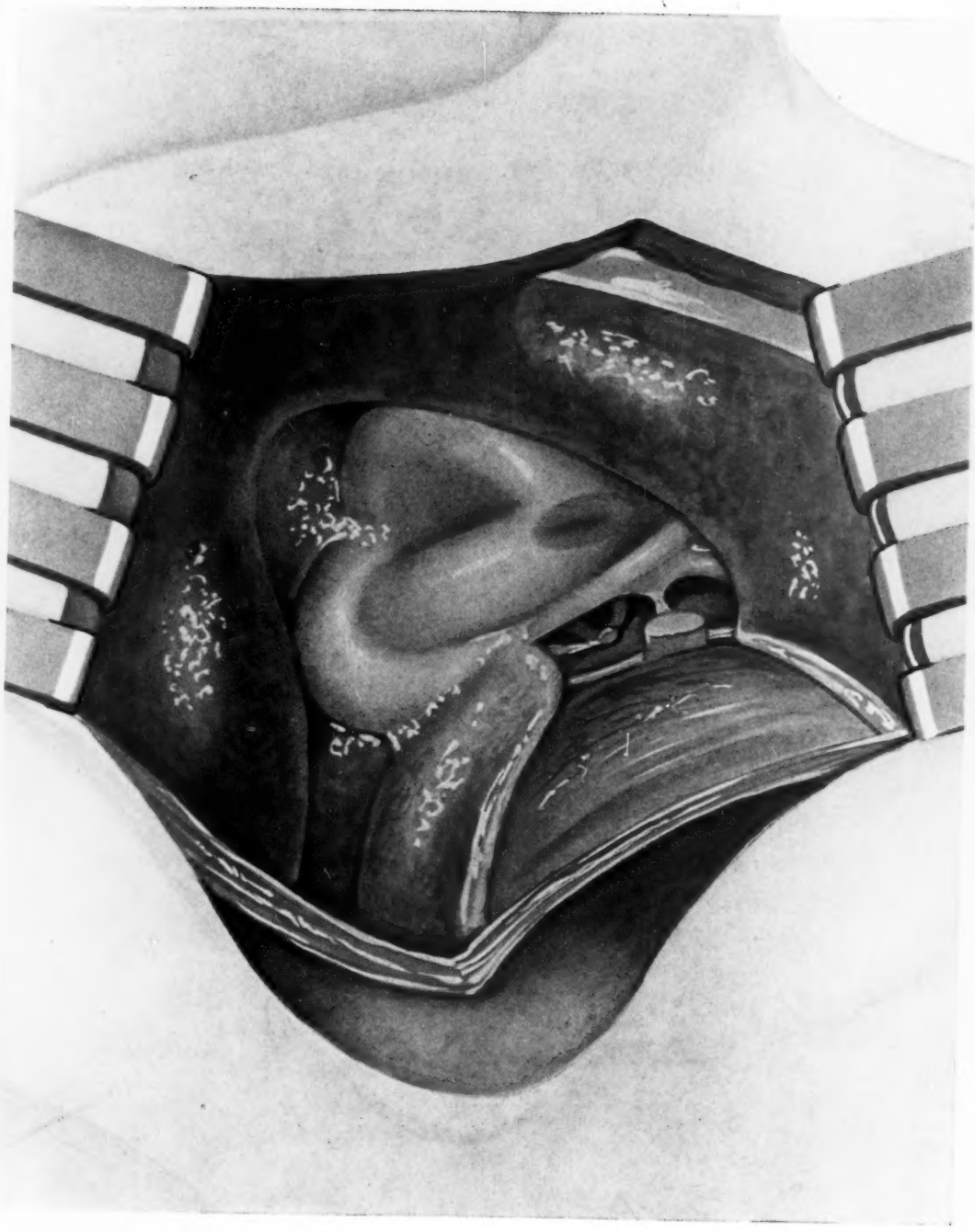

Fig. 21.—Creation of the fenestra nov-ovalis—step 1.

Fig. 22.—Creation of the fenestra nov-ovalis—step 2.

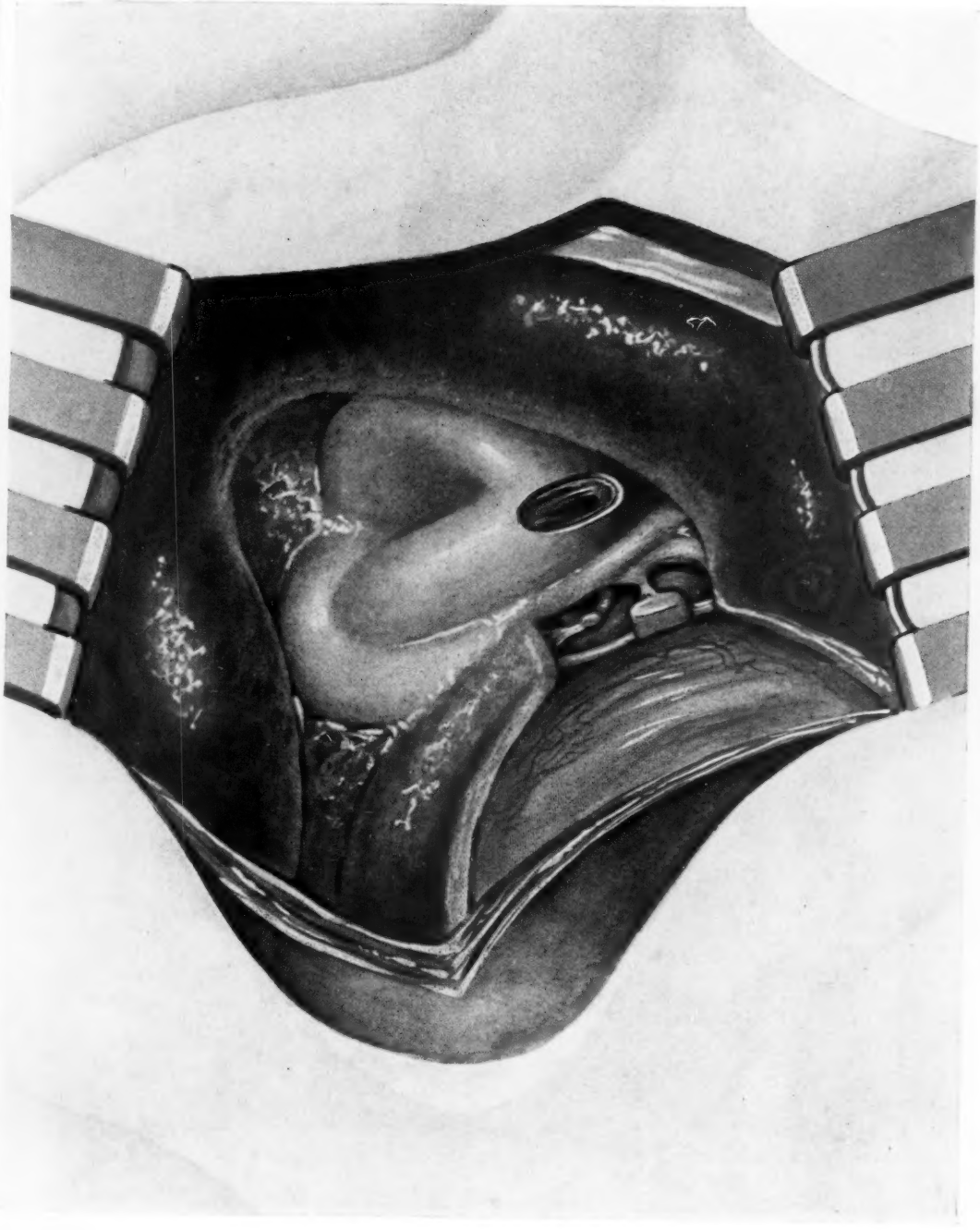

Fig. 23.—Creation of the fenestra nov-ovalis—step 3.

Fig. 24.—Creation of the fenestra nov-ovalis—step 4.

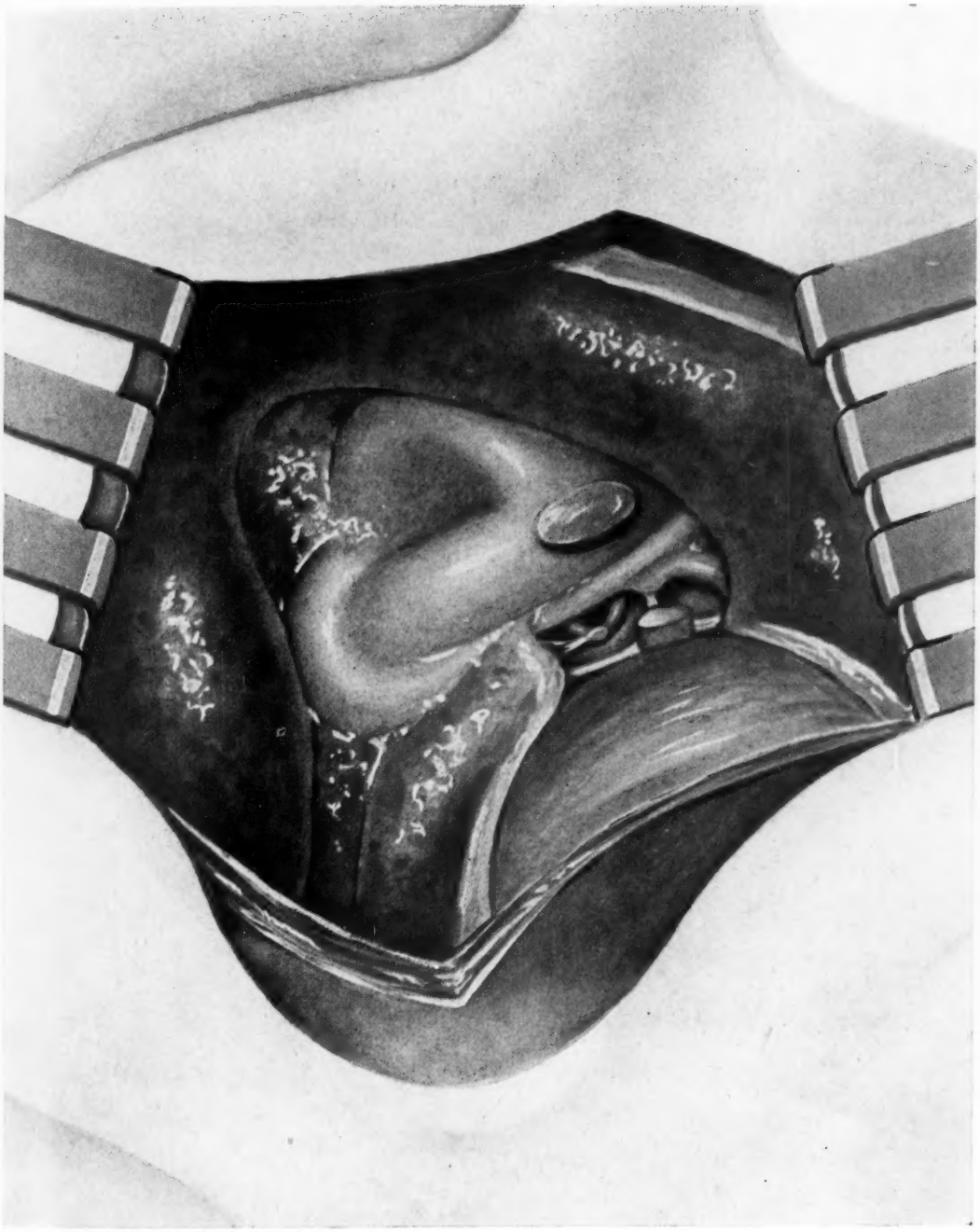

Fig. 25.—The mobile cartilaginous stopple is seen in situ within the fenestra nov-ovalis.

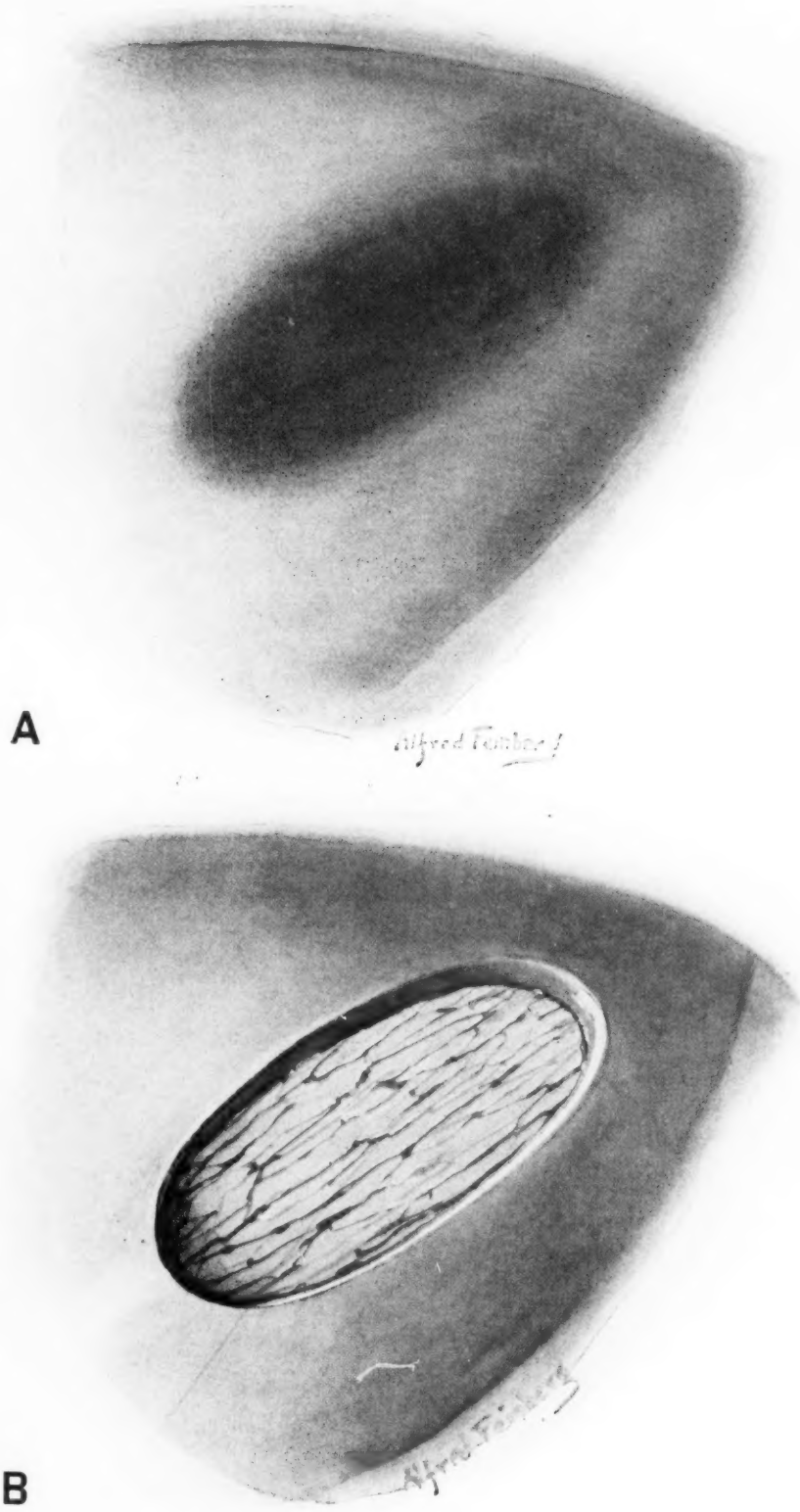

Fig. 26.—*A*, creation of the fenestra nov-ovalis—step 1. The area of the bony capsule in the surgical dome of the vestibule is worn down with a dental plug finishing burr until the endosteal layer is thinned to a transparency through which the perilymphatic space is visible.

B, step 2. The thinned transparent layer of the bony capsule has been pulverized and shredded and is seen lying on the endosteal membrane lining the perilymphatic space.

Fig. 27.—*A*, creation of the fenestra nov-ovalis—step 3. The pulverized and shredded endosteal layer of the bony capsule has been carefully removed, exposing to view the transparent endosteal membrane through which the endolymphatic labyrinth can now be visualized within the perilymphatic space.

B, step 4. The endosteal membrane has been removed, opening the perilymphatic space for a direct view of the endolymphatic labyrinth.

Fig. 2
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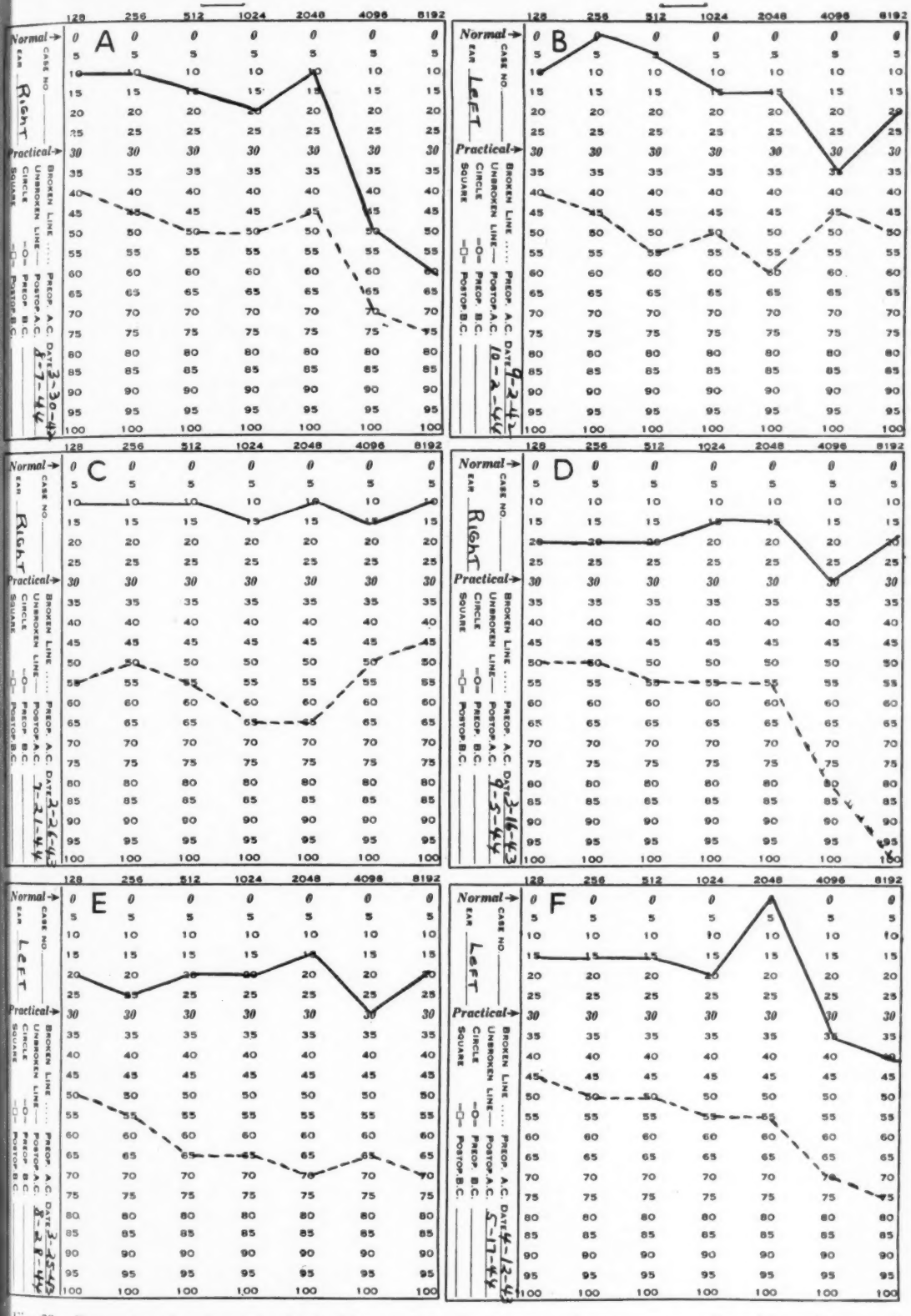

Fig. 28.—Examples of end results obtained by creating the Lempert fenestra nov-ovalis with mobile stopple: two years and five months after operation; *B*, two years after operation; *C*, one year and five months after eration; *D*, one year and six months after operation; *E*, one year and five months after operation; *F*, one year one month after operation.

but rather by closing it with a mobile cartilaginous stopple which closest resembles nature's way of keeping the normal fenestra ovalis mobile by closing it with the footplate of the stapes.

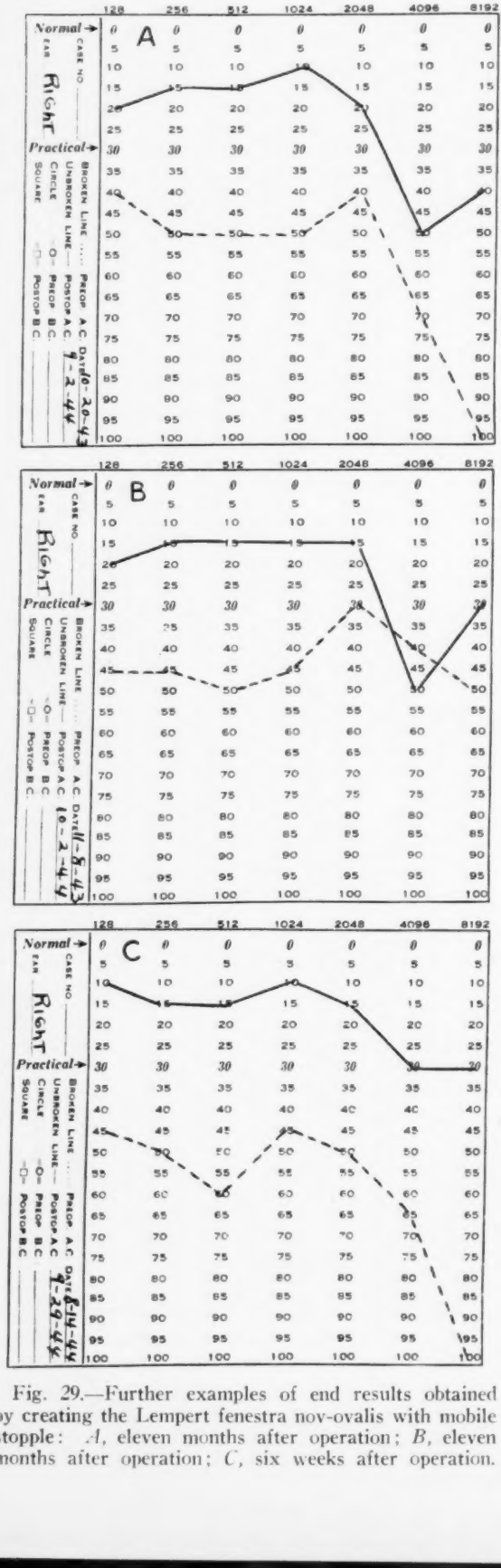

Fig. 29.—Further examples of end results obtained by creating the Lempert fenestra nov-ovalis with mobile stopple: *A*, eleven months after operation; *B*, eleven months after operation; *C*, six weeks after operation.

IS THE "AVERAGE DECIBEL GAIN" METHOD OF COMPUTING AND REPORTING SUCCESSFUL END RESULTS A TRUSTWORTHY ONE?

A careful analysis and study of my end results in 1,000 cases in which fenestration of the bony labyrinth has been performed during the last seven years revealed that:

The average decibel gain method of estimating and reporting end results following the fenestration operation, which is employed by some otologists and which consists in adding the postoperative decibel gains of the three conversational frequencies 512, 1,024 and 2,048 and dividing the sum total by three, is erroneous and often misleading to those interested in the surgical treatment of otosclerosis and to members of the

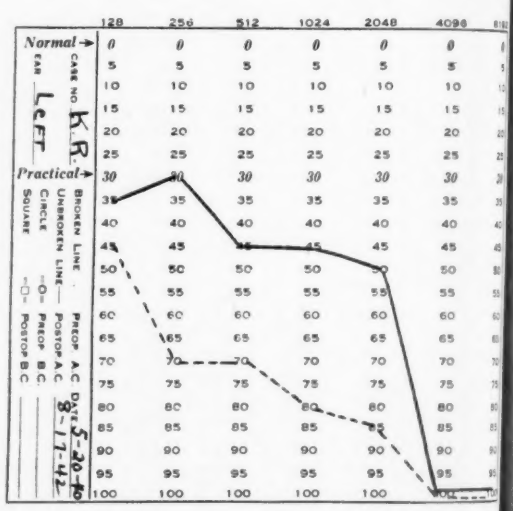

Fig. 30.—Audiogram of K. R. Although the patient has obtained a 32 decibel average improvement for the three conversational frequencies, she is nevertheless deaf for all practical purposes.

medical profession at large. Lumping all cases in which an average hearing gain for the conversational speech frequencies of 10 decibels and over was obtained following fenestration and reporting them all as showing successful, desirable end results prevent any correct and true evaluation of this form of surgical therapy. The average decibel hearing gain thus computed and reported as representative of a successful end result does not necessarily mean that the patient who now has this average hearing gain of 10 decibels or over is no longer deaf for all practical purposes or hears conversation better than preoperatively. The conduction deafened patient with a 45 decibel loss of hearing for the vital speech frequencies seeks medical care and with this degree of loss would be advised to have the fenestration operation. If this patient with a 45 decibel loss seeks relief, why should a patient

whose hearing has been improved from a 75 decibel to a 45 decibel loss for all the speech frequencies by fenestration have serviceable hearing?

The postoperative audiogram of a case taken at random out of my series will serve to illustrate the erroneous impressions created when all cases showing a postoperative average hearing gain of 30 decibels or over are reported jointly as the cases of patients successfully operated on (fig. 30).

HOW SHOULD END RESULTS BE COMPUTED AND REPORTED?

Experience has taught me that an end result should not be reported as finally successful until at least one year after operation and only when, at the end of that year, the hearing improvement has failed to show any continuous inclination to drop below the best obtained postoperative level. Once the hearing begins to drop, it usually drops to the preoperative level sooner or later, that is within six months, eight months or a year. If the hearing has not begun to show signs of receding by the end of one year, it is certain that either postoperative repair of the newly created fenestra has not taken place or that the postoperative process of repair which the fenestra has undergone has exhausted itself before closing the fenestra, thus leaving a permanent window which is large enough to permit the degree of mobility of the perilymph for air-borne sound which gives continuous practical, serviceable hearing.

In order to permit a true evaluation of this surgical treatment for otosclerosis, the end results obtained with this operation should be reported as follows:

1. If the hearing was improved to the 30 decibel level or higher for the conversational frequencies 512, 1024 and 2048 and maintained at that level of improvement for at least twelve months postoperatively, allowing only for the normal variable of 5 decibels of audiometric testing, the result should be reported as follows: Complete rehabilitation for social and economic contacts as a result of restored hearing was accomplished in ..... cases.

2. If the hearing was improved but the improvement did not reach the practical 30 decibel level in the 512, 1024 and 2048 frequencies, the result should be reported as follows: Partial rehabilitation for social and economic contacts as a result of improved hearing was accomplished in ..... cases.

3. If the hearing was not improved, the result should be reported as follows: The hearing was not improved and remained at the preoperative audiometric decibel level in ..... cases.

4. If the hearing became worse in the operated ear, the result should be reported as follows: Audiometrically, the hearing was depressed to a decibel level lower than the preoperative level in ..... cases.

By universally adopting and adhering to this method of reporting end results, otologists can be assured that though the quantitative picture of successful end results which will be obtained with this method of reporting will not be as bright looking as reports of end results based on the average decibel gain method of computing and reporting could paint it, even the staunchest opponents of this work will find it bright enough to become converted to it.

WHAT ARE THE DANGERS ACCOMPANYING THIS OPERATION?

There is no special danger to life or limb involved in a patient's submission to this type of operation. However, the operation is not accompanied by any special immunity to untoward incidents. Occasionally embolism or coronary thrombosis occurs during the period of convalescence following any major or even minor surgical intervention. The fenestration operation has not proved itself to be free from such unforeseen sequelae.

IS THERE DANGER OF INJURY TO THE FACIAL NERVE AS A RESULT OF THIS FORM OF TREATMENT?

With the facial nerve exposed to direct view, there is no reason why this nerve should ever be injured except accidentally. However, transient facial paresis may occur occasionally. This results from a postoperative inflammatory reaction of the soft tissues covering the region of the facial nerve. From absorption of secretions from the postoperative wound through the thinned-out bony facial canal, occasionally perineuritis results. This usually makes its appearance anywhere from one day to two weeks postoperatively and, as a rule, completely disappears within a period varying from ten days to three weeks after onset.

IS THERE ANY DANGER FROM POSTOPERATIVE INFECTION FOLLOWING THIS OPERATION?

Postoperative infection is not one of the sequelae to be feared as a result of this surgical procedure and is rarely encountered, because one is dealing with an infection-free, sterile operative domain and employing every extreme aseptic precaution for keeping the surgical terrain from becoming secondarily infected. But that does not necessarily preclude the possibility of an infection occurring postoperatively on rare occasions which are beyond one's control and totally unavoidable. This is as true of this branch of surgery as it is of operations on the brain or the eye or of abdominal operations where no

infection exists preoperatively but whence infection follows, nevertheless, postoperatively, only it follows more rarely in fenestration surgery.

IS THERE ANY DANGER OF IMPAIRING THE HEARING FURTHER WITH THIS OPERATION?

The endolymphatic labyrinth and the organ of Corti are undoubtedly the most delicate anatomic structures in the human body. The resistance of these structures to exposure and surgical trauma varies greatly, and in some persons it is low. With every possible aseptic precaution taken and with the greatest amount of surgical care exercised to traumatize the endolymphatic labyrinth as little as possible, postoperative sterile inflammation of the labyrinth will occur and occasionally result in damage to the organ of Corti with further loss of hearing. When one considers the fact that clinical otosclerosis is a hopeless progressive form of deafness which usually results in total loss of hearing, a further loss of hearing resulting from an attempt to improve the hearing is relatively not such a tragedy as it would at first glance appear.

Neither the accidental loss of vitreous occurring during extraction of a cataract nor the loss of an eyeball from infection, and not even the occasional additional loss of sight in the other eye, resulting from sympathetic ophthalmia, with consequent total irreparable blindness following operation for a cataract, has discouraged the ophthalmologist from employing surgical methods of dealing with cataract successfully, or the blind from seeking restoration of their vision by submitting to this form of surgical treatment. They act despite the common knowledge that such unfortunate consequences, though not frequent, are nevertheless within the realm of possibility even when this surgical intervention is most carefully and most skilfully performed.

END RESULTS IN ONE THOUSAND EARS OPERATED ON DURING THE LAST SEVEN YEARS

Analysis with Respect to Whether Preoperative Hearing by Bone Conduction Was Good or Poor.—During the last seven years fenestration was performed in 1,000 cases. In 815 of these, the preoperative diagnosis was clinical otosclerosis with good hearing for bone-conducted sound. The results obtained in these 815 cases may be stated as follows: Complete rehabilitation for social and economic contacts as a result of restored hearing was accomplished in 571 cases. Partial rehabilitation for social and economic contacts as a result of improved hearing was accomplished in 92 cases. (The older people in this group, who did not have to earn a livelihood, seemed to be satisfied with such a result.) The hearing remained unimproved in 118 cases. The hearing was further impaired in 34 cases.

In 185 cases the preoperative diagnosis was clinical otosclerosis with poor hearing for bone conducted sound, suggestive of advanced degeneration of the cochlear nerve. The results obtained in these 185 cases were as follows: Complete rehabilitation for social and economic contacts as a result of restored hearing was accomplished in 42 cases. Partial rehabilitation for social and economic contacts as a result of improved hearing was accomplished in 39 cases. The hearing remained unimproved in 93 cases. The hearing was further impaired in 11 cases.

Of the 1,000 patients, 384 were operated on not less than three years ago and it is now anywhere from three to seven years since the operation. Of these 384 patients, 309 had good bone conduction, while 75 had poor or no bone conduction. Of the 309 with good bone conduction, 187 had practical, serviceable hearing restored and continuously maintained from three to seven years postoperatively. In 37 patients hearing was improved and maintained continuously from three to seven years postoperatively but did not reach practical level. In 65 patients the hearing was not improved. In 20 patients the hearing was further impaired.

Of the 75 patients with poor or no bone conduction, 12 had practical, serviceable hearing restored and continuously maintained. In 1 patients hearing was improved but not practical. In 42 patients hearing was not improved. In patients hearing was further impaired.

Six Year Results.—Of the 1,000 patients, were operated on over six years ago. Of these 24 have retained their practical, serviceable hearing for more than six years since operation and continue to hear for all social and economic contacts. One of these patients has now had this type of improvement for seven years. Thirteen patients have retained their improved though not practical hearing for more than six years.

Closures.—Revision in cases in which closure of the newly created fenestra was suspected has revealed complete or partial closure in 140 of the 1,000 cases. Eighty-eight of these closures took place in the first 300 cases, in which the fenestra was created in the prominence of the external semicircular canal. Fifty-two closures took place in the last 700 cases, in which the fenestra nov-ovalis was created in the surgical dome of the vestibule.

Analysis of 50 Consecutive Cases in Which Fenestration Was Done More Than Four Years Ago.—In 50 consecutive cases of clinical otosclerosis in which this operation was performed

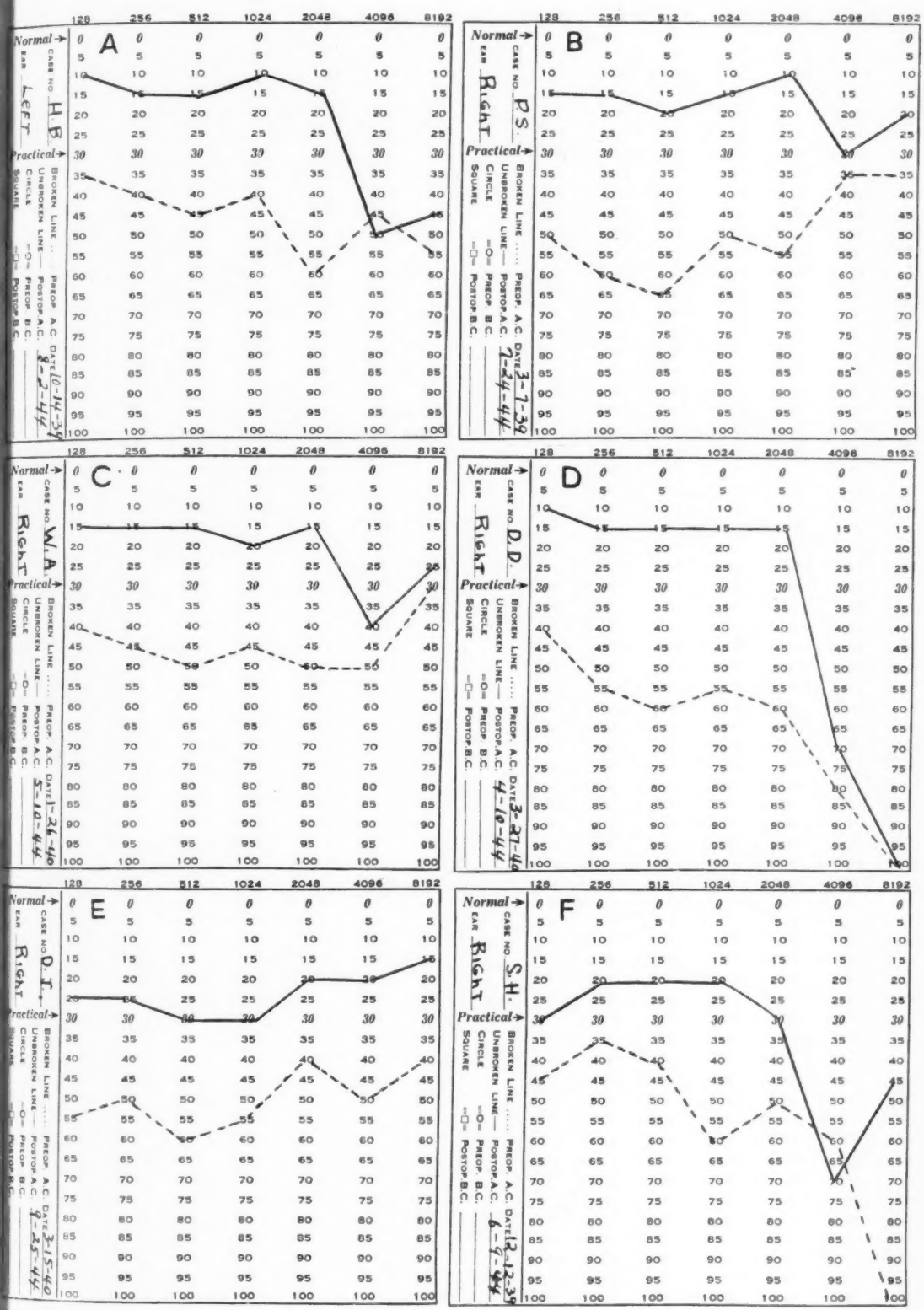

Fig. 31.—*A* to *F*, audiograms of 6 patients showing improvement five years after operation.

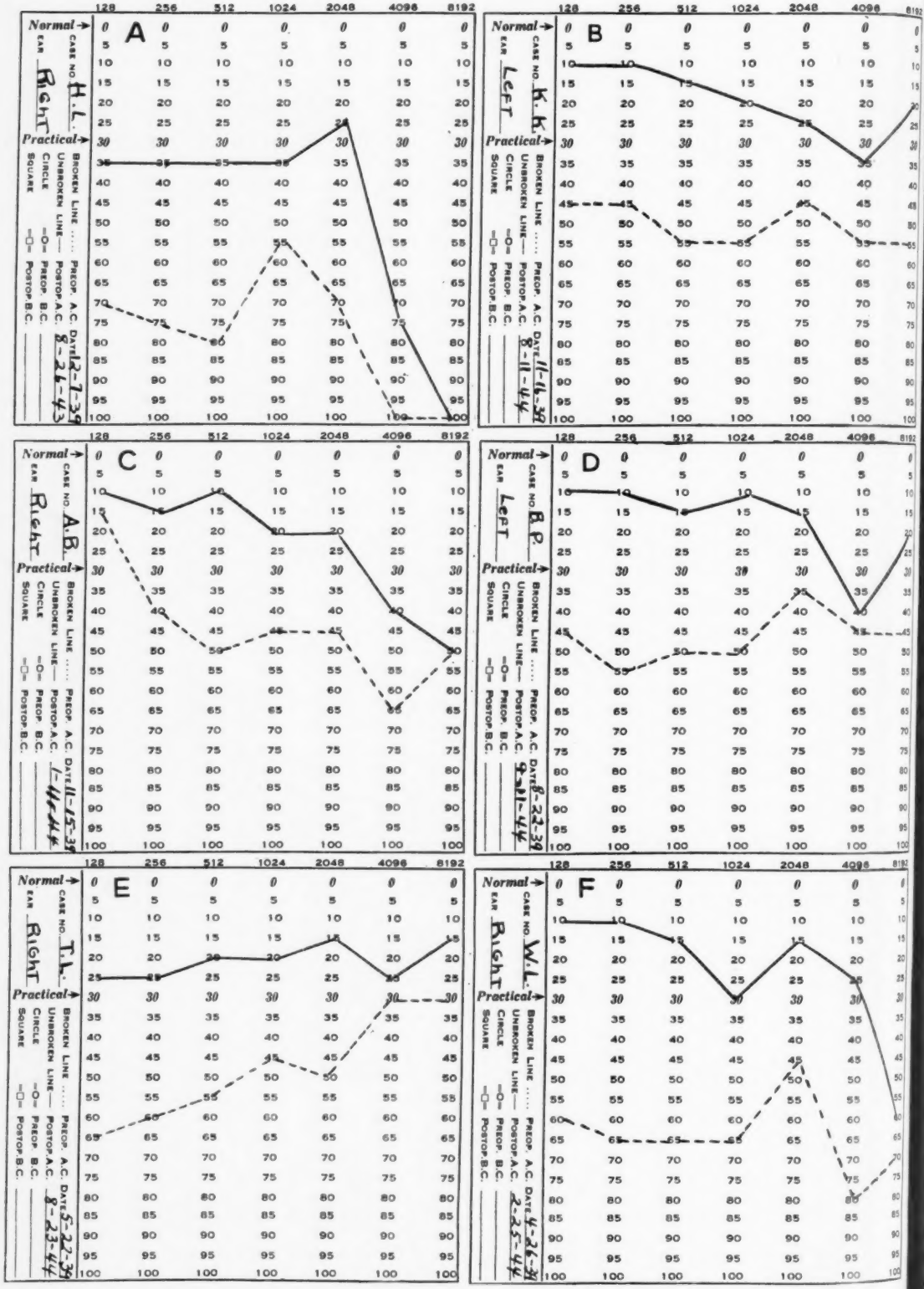

Fig. 32.—*A*, *B*, *C* and *D*, audiograms of 4 patients showing improvement five years after operation. *E* and *F* audiograms of 2 patients showing improvement five and one-half years after operation.

m the period beginning Nov. 15, 1939 and nding May 7, 1940 the following results were btained: Complete rehabilitation for social and conomic contacts was accomplished in 31 cases. artial rehabilitation for social and economic ntacts was accomplished in 7 cases. The hearng remained unimproved in 8 cases. The hearng was further impaired in 4 cases.

In 3 of the 50 patients complications and equelae were encountered as follows: One patient died two weeks postoperatively as a result of acute coronary thrombosis. Another patient, three weeks after operation on the left ear, showed signs and symptoms suggestive of an abscess of the left temporosphenoidal lobe. This ruptured into the ventricles one week after the onset of symptoms. The abscess was located and surgically drained. The patient died twelve hours after this intervention, although he had regained full consciousness. A round window graft had been inserted in the same left ear by Dr. Hughson one year prior to the patient's submission to the fenestration operation. In a third patient thrombosis of the lateral sinus developed with abscess of the jugular bulb, suppurative labyrinthitis and acute suppurative meningitis of streptococcic origin. From cultures of both the blood and the spinal fluid positive growths of Streptococcus haemolyticus were obtained. I ligated and resected the jugular vein, drained the jugular bulb, performed labyrinthectomy and administered a sulfonamide compound. The patient recovered. Total loss of hearing in the ear operated on resulted.

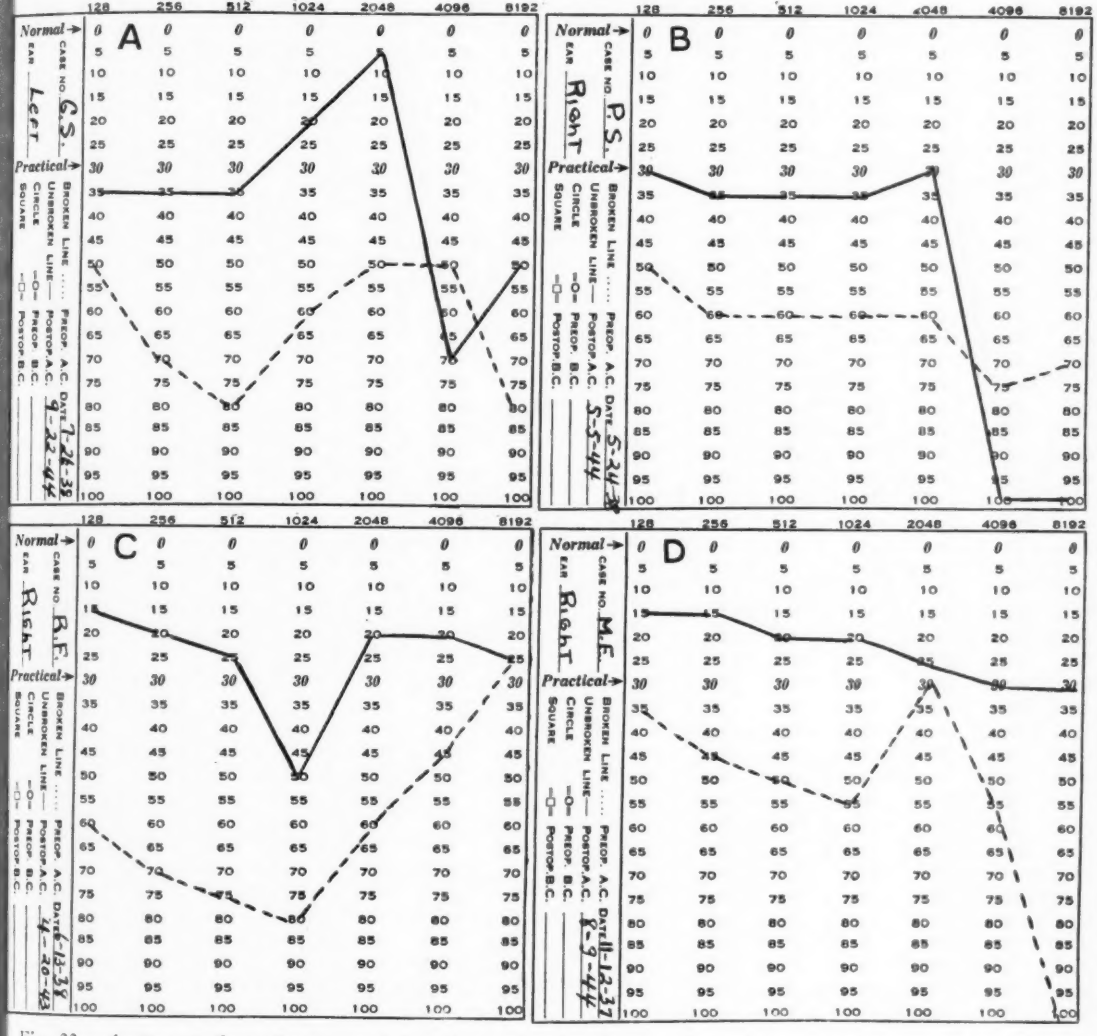

Fig. 33.—*A*, *B* and *C*, audiograms of 3 patients showing improvement six years after operation. *D*, audiogram of a patient showing improvement seven years after operation.

In 9 of these 50 cases revisions were performed. Complete rehabilitation for social and economic contacts as a result of restored hearing was accomplished in 3 of the 9 cases. The hearing remained unimproved in 6 of the 9 cases.

Complications and Sequelae in the 1,000 Cases in Which Fenestration Was Done.—Two patients died of acute coronary thrombosis within two weeks after operation. One patient died four weeks postoperatively from abscess of the left temporosphenoidal lobe.

One patient showed symptoms and signs suggestive of embolism in the left temporosphenoidal lobe immediately after operation. A diagnosis of abscess of the left temporosphenoidal lobe was eventually made, and the abscess was drained without disturbing the postoperative fenestration field. The patient made an uneventful recovery. Practical, serviceable hearing was restored and has been maintained ever since.

In 1 patient, as described in the foregoing section, thrombosis of the lateral sinus developed with abscess of the jugular bulb, suppurative labyrinthitis and acute suppurative meningitis of streptococcic origin. From cultures of both the blood and the spinal fluid positive growths of Str. haemolyticus were obtained. I ligated and resected the jugular vein, drained the jugular bulb, performed labyrinthectomy and gave a sulfonamide drug. The patient recovered. Total loss of hearing in the fenestrated ear resulted.

Transient facial paresis resulting from postoperative inflammatory perineuritis occurred in 14 cases. All except 1 of the patients recovered completely within periods ranging from two to three weeks following the onset.

The facial nerve was never injured in any of the 1,000 cases.

WHY OPERATE, SINCE OTOSCLEROSIS IS PROGRESSIVE AND INCURABLE?

It is true that the pathologic lesion known as otosclerosis is not yet a curable one. I, however, firmly believe that air conduction deafness which the otosclerotic lesion causes when it encroaches on the oval window and immobilizes the footplate of the stapes is not incurable. The cure of otosclerosis as a lesion may well be a biologic or a biochemical problem. The restoration of practical, serviceable hearing in clinical otosclerosis is, however, a purely surgical problem. After deafness for air-conducted sound occurs in otosclerosis, it keeps on becoming worse because the otosclerotic tumor keeps on growing and immobilizing the footplate of the stapes further and further, thus interfering more and more with the transmission of air-borne sound to the inner ear. Once the fenestra novovalis has been created in the surgical dome of the vestibule, where the otosclerotic lesion was never known to occur, it replaces nature's functionally impeded fenestra ovalis, and practic serviceable hearing is physiologically resume It no longer matters now whether the otoscler lesion continues to extend and the ankylosis the stapes progresses still further, because e since a new window was created the degree reestablished hearing has not been influenced the progression of the tumor in the region of t oval window.

I believe that the loss of hearing in o sclerosis is the direct result of a mechanical i pedance to the transmission of air-borne sou through the oval window to the organ of Cor as the loss of vision in cataract is the result the mechanical impedance to the transmission light through the lens to the optic nerve.

Air conduction deafness resulting from s pedial fixation in otosclerosis, if seen before t organ of Corti has become seriously damage can be cured by sidetracking the impeding lesi without curing the otosclerosis itself or remov its cause, just as blindness resulting from ca racts when not accompanied by degeneration the optic nerves can be cured by removing t impeding cataracts without removing the cau

There exists abundant evidence today, af the end results of fenestration have been watch for seven years, that continued progression the air conduction loss of hearing which characteristic of deafness resulting from stape fixation in otosclerosis and the progressive s ondary changes in the cochlear nerve acco panying it do not take place in any of the su cessfully fenestrated ears. That it is reasonab to assume this to be a direct result of the surgi treatment can only be deduced from the obse vation that the hearing in the ear not operat on continues to follow its unfavorable cour of both middle and inner ear depreciation.

The reason for the cessation of the progressi of the secondary loss of cochlear nerve funct is not yet clear and must be studied, but t should not be surprising, because neither a the reasons for the continuously progressive lo of cochlear nerve function in otosclerosis de and as yet established.

It is possible that changes in the cochle nerve in clinical otosclerosis are secondary or to the air conduction deafness which was pr cipitated by the spread of the otosclerotic tum in the region of the oval window and are n produced by the otosclerotic tumor per se. this is true, then by mobilizing the perilymp and the endolymph and thus doing away wi the air conduction deafness, one is removing th factors which could result in secondary dege eration of the organ of Corti.

WHY OPERATE FOR OTOSCLEROSIS IN THE PRESENT STAGE OF DEVELOPMENT OF THE HEARING AID?

Although I was never given the choice by the 1,000 patients on whom I have operated for clinical otosclerosis to decide for them between the fenestration operation and the hearing aid, I must admit that if I had been given the opportunity to choose for them, I should have strongly recommended the fenestration operation in preference to the use of a hearing aid. As a matter of fact, a majority of these patients were already wearing a hearing aid when seeking surgical relief for their hearing deficiency. For me to have refused them surgical relief of their deafness, which is the result of a mechanical impedance to air-borne sound, and to have insisted on their continuing to wear the electrical aid would have been tantamount to the orthopedic surgeon's refusing to operate for a surgically correctable physical infirmity causing inability to walk and recommending the use of crutches instead.

Today, with indisputable ability to restore physiologic hearing in a large percentage of cases and after having already maintained such results for periods as long as seven years, otologists need no longer hesitate to recommend the fenestration operation in suitable cases of clinical otosclerosis instead of the use of the hearing aid. Especially so, when they know that the hearing aid, when worn by a patient with this disease lulls the patient into a false sense of security which is bound to be followed by a sudden awakening to the fact that the formerly useful aid ceases to be an aid and that the one time opportunity for regaining and continously maintaining physiologic hearing by fenestration was missed by wearing this aid and is never to return.

Besides the foregoing scientific arguments for the fenestra nov-ovalis with mobile stopple and against the hearing aid in cases of clinical otosclerosis, the best practical argument is the fact that patients who were operated on successfully will tell one that the benefits they had obtained by the use of a hearing aid cannot at all be compared favorably to the advantages they now enjoy as a result of the fenestration operation.

No patient ever succeeded in rehabilitating himself completely or sufficiently either socially or economically by wearing an aid, which only serves to stigmatize him and to emphasize his deafness.

SUMMARY AND CONCLUSIONS

One thousand patients with clinical otosclerosis have been treated by fenestration of the labyrinth over a period of seven years. In 300 of these patients the external semicircular canal was fenestrated, while in the last 700 the fenestra nov-ovalis was created in the surgical dome of the vestibule. The end results obtained in these patients are reported. Practical, serviceable hearing for all social and economic purposes was restored and continuously maintained in a large percentage of the patients.

Closure of the newly created fenestra by new bone formation and damage to the organ of Corti as a result of serous labyrinthitis were found to be the two major obstacles to the still greater success of this surgical treatment.

A research study of the aforementioned two major causes of defeat in the surgical therapy of clinical otosclerosis was made in the 1,000 cases in which fenestration was done. As a result of this study it was found that it is not necessary to keep the newly created fenestra continuously open in order to maintain the hearing improvement obtained following fenestration, provided the closure can be kept mobile.

The Lempert fenestra nov-ovalis with a mobile stopple was evolved. This is a new technic based on a surgical principle different from any heretofore employed for the improvement of hearing in clinical otosclerosis. The newly created fenestra is immediately closed with a mobile cartilaginous stopple which simulates in function the normal stapedial footplate.

I am now employing this technic exclusively, because I am convinced that time and a large number of cases will eventually prove that both major causes of defeat, encountered with all previously employed technics which were based on the principle of keeping the fenestra open, can thus be conquered.

The principle of employing a mobile stopple to close the newly created fenestra opens new vistas for the still further simplification of the technic for the improvement of hearing in clinical otosclerosis. Various methods and means of accomplishing such simplification are now being investigated.

RECONSTRUCTION OF THE FACIAL NERVE

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Reconstruction of the facial nerve from the point of view of the otologist implies a restoration of its function by surgical means. In the main two procedures are advocated, the grafting of a nerve transplant and the end to end anastomosis. Both have been the subject of careful scientific research which has broadened knowledge of nerve regeneration immeasurably and created an epoch in the progress of surgery.

Fundamental principles have been evolved which are now at the disposal of the surgeon and when put to practical use give rise to results of a most gratifying character. While basic concepts are fairly well formulated and definitive technics established, much experimental work is now in progress which gives promise of improving and refining the methods which have been given avowed acclaim. As in many other fields of medicine, the present world conflict has made diligent study of the restoration of nerves imperative, and much knowledge of great practical value will undoubtedly come from the laboratories now at work on this problem.

The discussion contained herein is of necessity limited to two objectives, first, a plea for the preservation of the facial nerve and, second, a recommendation that the end to end anastomosis be used if practicable when the nerve is injured beyond its ability to recover spontaneously. Some one will ask: Why a reiteration of that which is evident? Still I venture to say that there are features of these texts which are worthy of more thoughtful consideration and emphasis than have been given them in the past.

To be specific, there is a feeling on the part of many surgeons that extensive operations on the parotid gland cannot be performed without sacrificing the facial nerve. It is argued that because the nerve passes through this structure it will of necessity be destroyed in any surgical procedure which calls for the total extirpation of the gland. The literature conveys this impression, and authoritative confirmation is given it by some of the textbooks.

Read at the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Cleveland, Jan. 12, 1944.

However, I should like to sound a m optimistic note by stating most emphatically t a complete resection of the parotid gland can accomplished without destruction of the fa nerve. This objective is by no means futile, despair of the preservation of the nerve is justified even when an extensive cancer of gland is exposed on surgical intervention. have encountered many mixed tumors of parotid gland with cancerous degeneration have operated on them successfully without p manent loss of function of the facial nerve. several instances, the fibers of the nerve w at first sight irretrievably lost in an infiltra neoplasm; nevertheless, by patient and pa taking dissection they were eventually preser for periods which would seem to connote clin cures.

When a tumor which obviously involves large part of the parotid gland is to be remov it is recommended that the first steps in t operation lead to an exposure and identificat of the nerve trunk. This, of course, is advis only when the size and the position of the tum definitely indicate that the lesion must embr the regions traversed by the important fibers the facial nerve and that its extirpation w apparently be accomplished by grave risk injury to the nerve. It is true that in many ca the mixed tumor of the parotid gland comme within the inner prolongation of the carotid l of the gland and lies inferior to the nerve b ments. A simple retraction of the latter upwa after the lesion has been exposed suffices to k them intact and preserve their function.

I offer for consideration the following surgi approach, a technic which lends itself admir to an exposure of the facial nerve near its e from the stylomastoid foramen with the mi mum risk and the maximum facility:

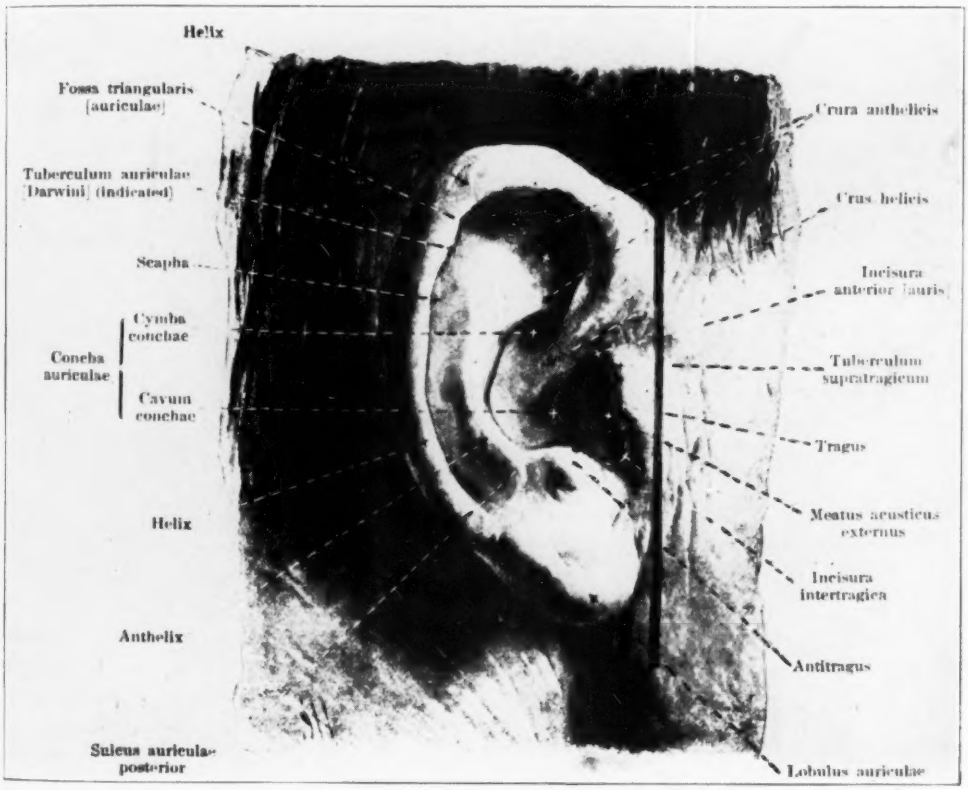
A vertical incision is made through the sk and the subcutaneous tissue immediately in fr of the tragus from the zygoma above to t angle of the jaw below (fig. 1). The lam of the tragus is thus identified, and the diss tion is carried inward with a submucous elev

ng the cartilage of the external auditory canal the direction of the styloid process. In gen- the route follows the anterior wall of the ternal auditory canal. The parotid gland is tly retracted forward.

The first important landmark to be identified he styloid process. Emerging from its mesial face near its base is the large main trunk of facial nerve enveloped by fascia but clearly ealed when the latter is teased away and dic stimulation applied to its fibers. After nerve has been discovered in this position, it traced forward into the parotid gland and efully delineated by painstaking dissection hin this structure (figs. 2 and 3).

branches which communicate with one another and with fibers of the fifth nerve to form a network which extends from the orbit above to the mouth below. The two main branches of the cervicofacial division are the mandibular and the cervical. The former may dip well below the margin of the mandible and curve superiorly again to supply the muscles of the lower lip and the chin. The cervical branch penetrates the superficial layer of the deep cervical fascia and gives off slender filaments which may descend to the level of the hyoid bone (fig. 3 *B*).

Embryologically it can be demonstrated that the parotid gland develops lateral to the main branches of the facial nerve. During the early



ig. 1.—External ear (reproduced from Spalteholz, Werner: Hand Atlas of Human Anatomy, edited and trans- d from the third German edition by Lewellys F. Barker, Philadelphia, J. B. Lippincott Co., 1900-1903). Note line of incision for exposure of the main trunk of the facial nerve where it emerges from the stylomastoid amen.

or clinical purposes interest centers in two n branches of the facial nerve, the superior temporofacial division and the inferior or vicofacial fibers. They constitute the two mary branches of the facial nerve which arate from the main trunk within the parotid nd, the former directed upward, the latter iquely downward toward the angle of the er jaw. It is to be remembered that the porofacial division divides into many

stages of its growth, however, the gland buds envelop the nerve to the extent that it finally becomes surrounded by glandular tissue. Thus it will be observed that the fibers of the facial nerve lie in the deeper structures of the parotid gland, near its mesial surface—an anatomic fact which lends a sense of security when surgical procedures within the gland are contemplated. In dealing with an abscess of the parotid gland one may cut deeply into the gland without grave

danger of injuring the nerve. A tumor or a cyst attaining considerable depth within the gland may be excised with little risk of severing important fibers. It is not uncommon, however, for a mixed tumor, which represents a teratologic lesion (misplaced or aberrant cells), to develop within the parotid tissues medial to the nerve and to push it laterally in the process of its growth. Under such circumstances one may find the facial nerves superficially placed and exposed to injury at the initial incision for the removal of the neoplasm of the parotid gland. All of which emphasizes the value of exposing the nerve near the stylomastoid foramen and tracing it forward from this point when a lesion of this character is suspected.

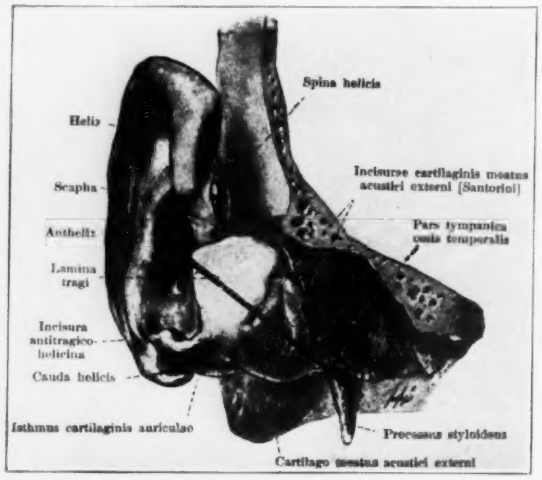


Fig. 2.—External ear and a portion of the temporal bone (reproduced from Spalteholz, Werner: Hand Atlas of Human Anatomy, edited and translated from the third German edition by Lewellys F. Barker, Philadelphia, J. B. Lippincott Co., 1900-1903). The dark line indicates the surgical approach to the facial nerve. After the lamina of the tragus has been identified, the dissection is carried along the anterior surface of the external auditory canal downward and mesially toward the styloid process. The nerve is easily identified, emerging from the mesial surface of the styloid process near its base. Faradic stimulation is helpful in locating the nerve.

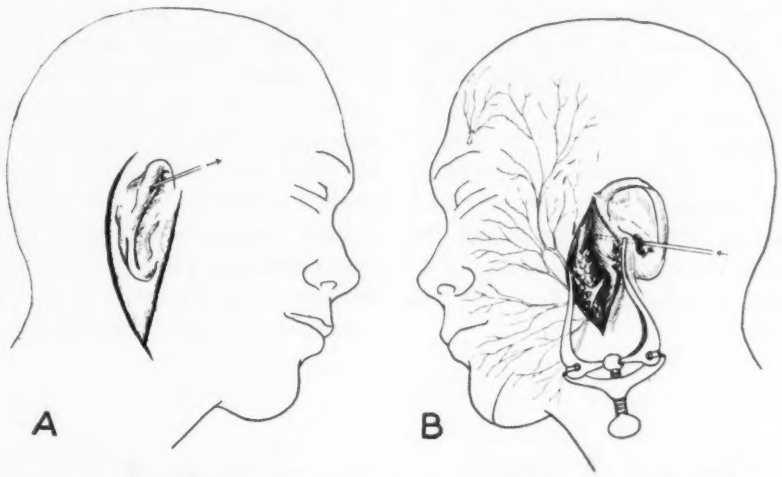
Returning to my original premise, the preservation of the facial nerve, I have attempted, in the foregoing discussion, to outline its important ramifications, make a plea for their identification and urge that they be given the respect which they command in operations on the parotid gland. As stated early in my remarks, it is perfectly possible and moreover practicable to remove the entire parotid gland with preservation of the critical fibers of the seventh nerve. Careful, painstaking and meticulous delineations of the nerve fibers, which may be identified by light faradic current, will frequently lead to results exceeding one's fondest hopes. The removal of an infiltrating neoplasm of the parotid gland is not necessarily an exception to the attainment of this ideal. It can be attained in an amazing degree by those who will operate in this region with patience, clarity of objective and precision of action.

The second objective of my text dealing with traumatic injuries of the facial nerve was the recommendation that an end to end anastomosis be used when practicable. This is predicated on the assumption that the nerve has been severed by a clean cut or that only a small part of it has been damaged. To be sure, there have been many accidents which have destroyed the nerve over its entire course within the mastoid process and middle ear so that the only hope of rehabilitation lay in the use of the nerve transplant or graft. This procedure has been most skillfully accomplished with admirable results by many members of the medical profession. There are times when this operation is absolutely imperative; when no alternative is at one's disposal. However, one must bear in mind that if a choice of procedure is permitted, the risk of failure is always greater when a nerve transplant is employed than when the two ends of the facial nerve are brought together in an end to end anastomosis.

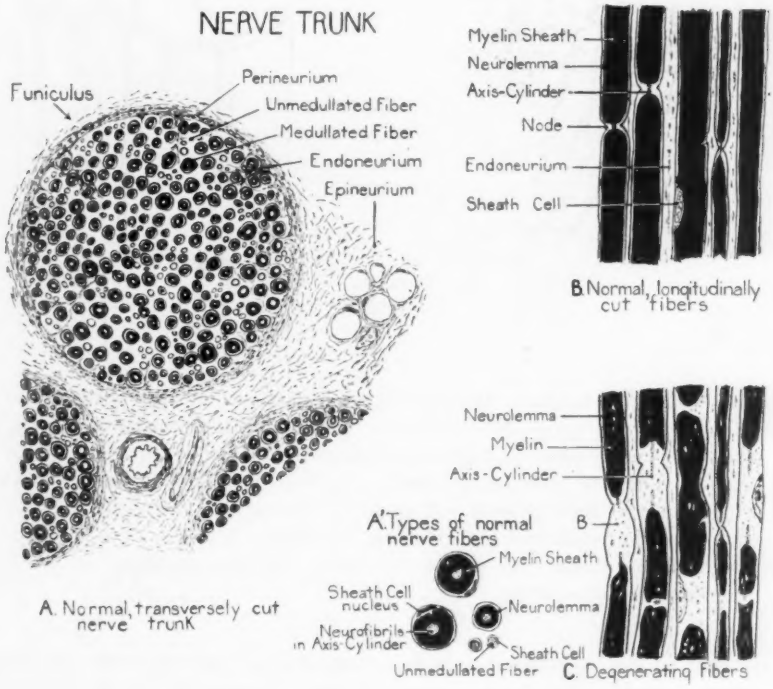
What happens to the facial nerve when it is severed (fig. 4)? The distal end degenerates over its entire extent, but the proximal end keeps on growing. The nerve fibers push forward and if they meet resistance in granulation tissue they criss-cross, wrap around each other in spiral fashion or grow otherwise in an inordinate arrangement to form what is termed a neuroma. If, however, one gives the neuraxes the pathways along which they may grow and prevents or removes any resistance to their progress, the end result is a complete regeneration of the nerve. It has been estimated that the average rate is probably a millimeter a day.

It goes without saying, therefore, that the end to end anastomosis offers less risk than the grafting of a nerve transplant and should be employed whenever practicable. The fresh ends of the severed nerve are brought together with avoidance of rotation so that the down growing neuraxes will extend distally within the neurilemma sheaths of the degenerated nerve. Care must be taken to approximate the nerve sheaths so that no crevice will remain at the joint which will permit ingrowth of granulation and con-

tissue. Next to infection, the latter occurce is probably the greatest deterrent to nerve eneration. Recent studies seem to indicate through and through sutures damage the nerve bundles and frustrate repair. Consequently, anastomoses are now being performed with the use of sutures which pass superficially through the sheath and connective tissue of the



ig. 3.—*A,* incisions employed when the facial nerve is rerouted in the temporal bone and relaxed by eating its branches forward within the parotid gland. *B,* exposure of the main trunk of the facial nerve e it emerges from the stylomastoid foramen. Once it has been identified in this region a delineation of bers within the parotid gland is made possible.



g. 4.—*A,* transversely cut normal nerve trunks. *A',* types of normal nerve fibers. *B,* longitudinally cut al fibers. *C,* degenerating fibers. When the nerve is severed, its distal end undergoes complete degenera- The myelin becomes segmented, the nuclei of the sheath cells disappear and finally the axis-cylinder dis- rates. The last part of the nerve to be destroyed is the neurolemma (sheath). It is present in a more or normal state two or three weeks after the nerve has been severed. Thus degenerated nerve transplants are chosen because they function as the best pathways along which regenerated nerve fibers may proceed. serve admirably as conduits or channels along which the neurofibrils will grow with the minimum of uption.

nerve. The use of a plasma glue (a biochemical preparation) is strongly recommended by recent workers in this field.

As has been stated, grafting may be the only method of choice when large portions of the facial nerve have been destroyed (fig. 5). This entails, however, two points of union and thus doubles the hazard of fibrous connective tissue encroaching on the joints. Moreover, the transplant may be attacked and devoured by leukocytes before it has served its purpose as a pathway along which neuraxes from the proximal end of the nerve may proceed. The longer the graft the greater is the chance of failure. Obviously, any degree of infection adds to the risk of its disintegration.

The radical mastoidectomy is first perform the point of injury located and the facial ne lifted out of the fallopian canal over its en extent. The rim of the stylomastoid foram is then removed so that the nerve can be fr at this point of emergence from the skull the desired distance within the parotid gla If the dissection is carried far forward along temporofacial and cervicofacial divisions, amazing degree of relaxation of the nerve obtained and end to end anastomosis acco plished within the mastoid bone without tens I have frequently estimated that as much a centimeter and a half of nerve was gained by t technic. Consequently, in the vast majority cases of nerve injury that have come to my att

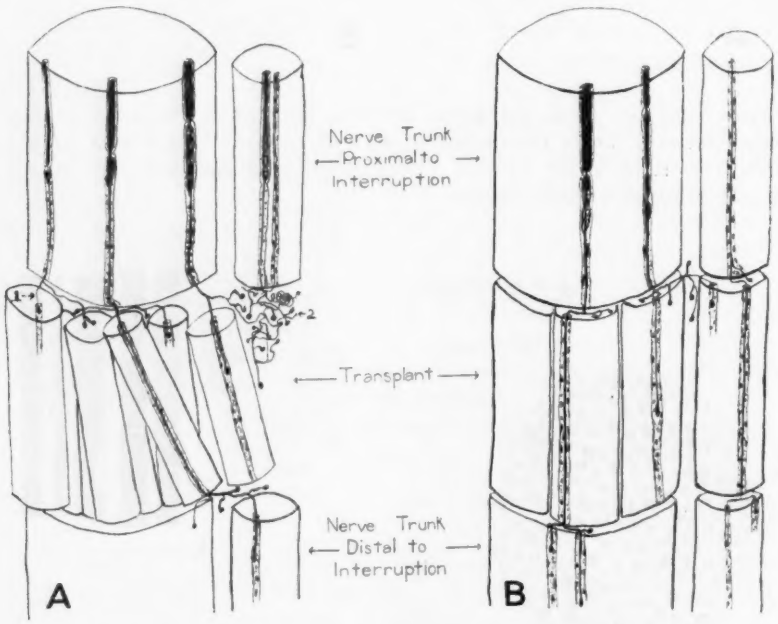


Fig. 5.—*A*, diagram showing a transplant rotated, angulated and out of line with the proximal and di ends of the cut nerve. Some of the neurofibrils find their way within the sheaths of the transplant (*1*); ot miss the pathway, wrap about each other and grow inordinately to form a neuroma (*2*). *B*, diagram show a nerve transplant in good alinement with nerve trunks, proximal and distal to the interruption. The budd neurofibrils find little resistance to their growth and progress throughout the nerve transplant. A succes technic in using a nerve transplant embraces the following principles: (1) the introduction of the transplan direct line with the proximal and distal ends of the cut nerve; (2) the avoidance of angulation and tors (3) a clean wound. Infection frustrates repair.

Now to get to the gist of this argument: It is my belief that too little attention has been given in the past to a wide exposure of the facial nerve and a rerouting of it when it has sustained injury within the mastoid bone. It is amazing the extent to which this nerve may be short circuited when lifted out of the horizontal and vertical portions of the facial canal and freed by extensive dissections forward within the parotid gland.

tion it has been possible to bring about an e to end anastomosis by the relaxing and reroute technic described.

I doubt that the feasibility and practicabil of this procedure have been accorded the recog tion which they deserve, and it is for this rea that I have taken the risk of pointing out t obvious, to discuss the second premise of th text.

SUMMARY

The facial nerve should be preserved in operations on the parotid gland. One need not despair of maintaining its fibers intact even when large infiltrating lesions of the gland are encountered.

An optimistic point of view in regard to safeguarding the facial nerve can best be maintained by an initial exposure of the trunk near its emergence from the stylomastoid foramen. Thus early identification of the nerve is made possible and its important ramifications may be traced with facility throughout the parotid gland.

The advantages of an end to end anastomosis over use of a nerve transplant seem obvious. The tortuous route of the facial nerve can be appreciably shortened by lifting the nerve out of its bony canal and freeing it along its course in the parotid gland. A union of the ends of a severed nerve which at first seemed futile may often be accomplished by this method.

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ACUTE NASAL ACCESSORY SINUSITIS COMPLICATED BY PURULENT MENINGITIS; RECOVERY

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Purulent meningitis following inflammation of the accessory cavities of the nose is a rare complication, and practically always it has a fatal issue. In 1927, in the course of my work under Prof. Robert Bárány in the otorhinolaryngologic department of the University Hospital, Uppsala, Sweden, I demonstrated a case in which recovery occurred after application of the same principles of treatment as were followed in the 2 cases to be described here. Professor Bárány told me at the time that he had never previously seen a similar case in which the patient had recovered, nor did he believe he had ever seen one described in the literature. Owing to circumstances beyond my control, a report of that case was never published. It tallied in all essentials with the 2 cases to be described.

Up to the time of the publication in *Acta oto-laryngologica* [1] and, later, in greater detail, in the *Journal of Medical Practice* [2] of my second case of recovery from streptococcic meningitis secondary to acute inflammation of the accesory cavities of the nose, no similar case had been published in the world's medical literature. In June 1943 I treated an almost identical condition in a third case in which recovery occurred. Before giving an account of this last case and of the treatment, I shall give a short description of the aforementioned published case. The circumstance that throughout their development the 2 cases were almost identical makes it probable that one deals with a disease in the course of which several important details were previously overlooked, with the consequence that the simple yet effective therapy was not resorted to in time and this may have contributed to the high mortality, the diagnosis probably having been faulty or incomplete.

The history of the case which I published at an earlier date was as follows:

On Dec. 18, 1929 a 15 year old schoolboy was sent to the hospital for patients with epidemic diseases in Göteborg, with the diagnosis "meningitis cerebrospinalis epidemica(?)" On the whole the patient had always been healthy otherwise. About ten days before admission to the hospital he had a cold in the head, which gave rise to increasing obstruction in the nasal cavities together with soreness and swelling of the nostrils. A week later he felt pains in the lower extremities and had a severe headache. On the days immediately following, he had fits of shivering, vomited and showed a rise of temperature. The day before admission he began to complain of stiffness of the neck and double vision.

On December 18 the pulse rate was 60 and the temperature 38 C. (100.4 F.). The general condition was relatively good. The psyche was calm, and there were no convulsions. There was considerable stiffness of the neck, and the trunk showed a slight degree of opisthotonos. The Lasègue sign was strongly positive bilaterally. There was paresis of the external rectus muscle of the right eye. Lumbar puncture showed that spinal fluid under a pressure of 230 mm. of water; 30 cc. was drawn off; the final pressure was 80 mm. As a measure of precaution 20.3 cc. of antimeningococcus serum was injected. The fluid was cloudy, and polymorphonuclear leukocytes were abundant. No bacteria were observed in the fluid.

On December 19 the pulse rate was 64 and the temperature 38.8 C. (101.8 F.). His condition was no worse, he was no longer psychically clear and complained of severe headache. He had difficulty in breathing through both sides of the nose. The nostrils were swollen, reddened and coated with crusts of dried secretion. Lumps of pus the size of hazelnuts were seen in each nasal vestibule. The mucous membrane in the nasal cavities was notably reddened and somewhat swollen; here and there were small aggregations of stringy purulent secretion. Over the central concha and its immediate surroundings on both sides was a thin, slimy purulent deposit. There was no discharge of pus. A rather large adenoid was noted in the epipharynx. Both the drum membranes were normal. On puncture and flushing of each maxillary sinus two to three lumps of pus the size of hazelnuts were recovered. The flushing fluid showed slight diffuse cloudiness. Lumbar puncture produced spinal fluid under a pressure of 290 mm.; 30 cc. was drawn off; the pressure was 100 mm. The fluid was cloudy, with masses of polymorphonuclear leukocytes. Culture disclosed streptococci in the lumbar fluid.

On December 20 the pulse rate was 64 and the temperature 38 C. (100.4 F.). The patient had slept peacefully the whole night. Headache had disappeared, but paresis of the external rectus muscle of the right eye and stiffness of the neck persisted. The Lasègue sign was positive. No flushing of the maxillary sinus was done this day, but the nasal cavities were pumped out a couple of times with Much's tube, with lumps of pus being recovered, especially from the right side.

On December 21 the pulse rate was 62 and the temperature 37.8 C. (100 F.). Stiffness of the neck decreased. Paresis of the external rectus muscle of the right and double vision were decreasing. When the maxillary sinus was flushed, the fluid was clou

1. Ebert, E.: Acta oto-laryng. **15**:104, 1931.
2. Ebert, E.: J. M. Pract., April 1936, vol. 1.

contained some small lumps of pus. Lumbar puncture showed a pressure of 240 mm.; 20 cc. was drawn off; the fluid was less cloudy than previously. Cultivation disclosed Streptococcus viridans and a gram-negative type of Diplococcus catarrhalis in the lumbar fluid.

On December 23 the pulse rate was 72 and the temperature was 37 C. (98.6 F.). The patient's condition had improved further. The fourth and last lumbar puncture was made. The pressure was 190 mm.; 20 cc. was drawn off; the final pressure was 80 mm. The fluid was slightly cloudy.

On December 29 the patient had no elevation of temperature, stiffness of the neck or paresis of the external rectus muscle of the right eye. The Lasègue sign was positive bilaterally at 45 degrees.

On Jan. 10, 1930 the patient left the hospital. The treatment had been as follows: flushing of both the maxillary sinuses was done every second or third day, as well as daily pumping out of the nasal cavities with Proetz's tube. After the patient's discharge from the hospital the flushing of the maxillary sinuses was continued as before, and on January 20 the fluid was still somewhat diffusely cloudy.

On January 22 there was ablation of the adenoid growths with the operative field under anesthesia induced with ethyl chloride. A few days later flushing of the maxillary sinuses was resumed, and on February 6 the fluid was clear. The patient felt quite recovered and reentered school.

The last case, observed in 1943, had the following history:

On June 1 a 21 year old conscript was admitted to the department of epidemic diseases of the Boden Garrison Hospital (physician in charge, Dr. A. Engel) with the diagnosis "meningitis cerebrospinalis epidemica(?)." The patient had always been healthy otherwise, was not aware of ever having the usual infantile illnesses, had not often had a sore throat, had never had ear trouble and had been vaccinated about a week before against smallpox.

About fourteen days before admission to the hospital the patient had a cold in the head for some days, but this did not trouble him much afterward. Instead he had a sore throat, a headache and a fever. These symptoms became worse day by day, and on May 30 a chill of shivering supervened.

The patient was admitted to the regimental hospital on May 31. His general condition was then somewhat affected, although he neither vomited nor had nausea. There was nothing to report from the internal organs. There was no stiffness of the neck, but the pharynx was reddened.

On the morning of June 1 his general condition rapidly deteriorated. He was pale, with clouded psyche, and had considerable stiffness of the neck. He was therefore transferred to the department of epidemic diseases of the garrison hospital.

On June 1 the pulse rate was 92 and the temperature was 37 C. (98.6 F). The general condition was greatly affected. For the most part the patient was quiet and somnolent but sometimes became unruly, wanted to get up, talked unintelligibly, groaned, complained of headache, and when examined moaned and made parrying gestures (after the patient had been restored to health he stated that he had not the faintest recollection of what had happened on that or the following day). He was obviously photophobic and had considerable stiffness of the neck. The body showed a high degree of opisthotonos. The Lasègue sign was positive bilaterally at about 60 degrees. The patellar and achilles reflexes were lively bilaterally. The Babinski sign was absent bilaterally. The pupils and pupillary reflexes were normal.

The blood pressure was 140 systolic and 70 diastolic. The sedimentation reaction was 52. A positive Mantoux reaction (0.10 cc. of diluted old tuberculin) measured 17 by 25 mm. The white corpuscle count was 28,000. Cultivation of the blood on plates and in bouillon gave no growth.

The drum membranes were normal; the tongue was dry and coated; the pharynx was rather strongly reddened; the tonsils were slightly enlarged, without exudation, spots or coatings; there were some small aggregations of dried secretion at the back of the pharynx as well as in the epipharynx; the epipharynx was otherwise normal. There was some deviation of the septum nasi toward the right, and a large crista was noted on the right side at the base of the septum. Small lumps of dried secretion were present in the nasal cavities. The mucous membrane in the nasal cavities was slightly reddened and swollen; here and there were small aggregations of stringy purulent secretion. There was no discharge of pus. On puncture and flushing of the maxillary sinuses three to four lumps of pus of the size of peas were recovered from each side. The flushing fluid also showed slight diffuse cloudiness. On direct examination of material from the nasal mucous membrane the Gram stain and methylene blue revealed no bacteria; secretion from the maxillary sinus when cultivated on ascitic fluid and dextrose bouillon gave a growth of staphylococci.

Lumbar puncture showed the spinal fluid under a pressure of 380 mm.; 45 cc. of milky fluid was drawn off; the final pressure was 110 mm. The Queckenstedt test indicated absence of any block in the vertebral canal. The result of the Pandy test was 4 plus; that of the Nonne test, 2 plus. The cell count was 22,375 leukocytes per cubic millimeter. The content of reducing substance was 0.020 per cent. The sugar content of the blood was 0.141 per cent. On direct examination of the spinal fluid no bacteria were observed. Cultivation of the fluid on ascitic fluid and dextrose bouillon disclosed no growth.

On June 2 the pulse rate was 72 and the temperature 37.4 C. (99.3 F.). The patient was somewhat better this day; he was bright and clear, although a little listless. The stiffness in the neck had decreased somewhat; it was possible to move the neck about more easily. When the maxillary sinuses were flushed, two to three lumps of pus, smaller than those obtained on the previous day, were recovered from each. The flushing fluid, too, was less cloudy. The urine was dark yellow, cloudy, acid. The Almén test demonstrated no albumin; the Heller test, traces. The Schlesinger test showed a weak reaction for urobilin. The specific gravity was 1.027. The sediment showed scanty white corpuscles, a single red cell and one granular cast. Lumbar puncture showed the spinal fluid under a pressure of 145 mm.; 11 cc. of cloudy fluid was drawn off; the final pressure was 100 mm. The result of the Pandy test was 3 plus; that of the Nonne test, 1 plus. The cell count was 5,325 leukocytes per cubic millimeter. No bacteria were found. Cultivation on ascitic fluid and dextrose bouillon gave no growth.

On June 3 the pulse rate was 64 and the temperature was 36.8 C. (98.2 F.). The volume of urine during twenty-four hours was 350 cc.

On June 4 the pulse rate was 68 and the temperature 36.8 C. (98.2 F.). The patient's condition continued to

improve. The white corpuscle count was 6,700. The urine during twenty-four hours totaled 500 cc.; it was dark yellow, cloudy, acid. The Heller test revealed no albumin. The sediment contained occasional white cells and one or two red cells. When the maxillary sinuses were flushed, one to two lumps of pus, smaller than those recovered the last time, were obtained from each side. For the rest the findings were the same as before.

On June 5 the pulse rate was 76 and the temperature was 36.5 C. (97.7 F.). The volume of urine during twenty-four hours was 1,000 cc. When the maxillary sinuses were flushed, no lumps of pus were recovered from either side. For the rest the flushing fluid was only slightly cloudy.

On June 6 the pulse rate was 80 and the temperature was 36.5 C. (97.7 F.). The volume of urine during twenty-four hours was 1,100 cc. The white corpuscle count was 7,000.

On June 7 the pulse rate was 76 and the temperature 36.3 C. (97.3 F.). The patient's condition continued to improve. The volume of urine during twenty-four hours was 875 cc. Lumbar puncture, the third and last, produced spinal fluid under a pressure of 140 mm.; 8 cc. of fluid, clear as water, was drawn off. The final pressure was 120 mm. The Queckenstedt reaction indicated no block in the vertebral canal. The Pandy test showed globulin (1 plus). The cell count was 74 per cubic millimeter, predominantly mononuclear leukocytes; red corpuscles were moderately numerous.

On June 8 the pulse rate was 72 and the temperature 36.6 C. (97.8 F.). The urine during twenty-four hours totaled 1,000 cc. and was yellow, slightly cloudy and acid. The Heller test demonstrated no albumin. The sediment contained an insignificant number of white cells. A slightly cloudy fluid was recovered when the maxillary sinuses were flushed.

On June 26 the patient left the hospital fully recovered. The treatment comprised repeated flushings, seven in all, of the maxillary sinuses—until the recovered fluid was perfectly clear—and rinsing of the nasal cavities every morning and evening with isotonic solution of sodium chloride (according to the method of Harke) up to and including June 15. The flushings were then discontinued, and the maxillary sinuses were afterward rinsed only once daily according to the method of Harke until the patient left the hospital.

Three lumbar punctures were made.

During the first six days the patient was given 0.5 Gm. tablets of sulfapyridine, one every six hours the first day and one a day the following five days.

As I have pointed out in my introduction, the 2 cases were almost completely identical with respect to the clinical picture as well as the course of the disease. After a short and slight prodromal stage, extremely serious symptoms of meningitis appeared, which soon broke down the patient both physically and psychically. These symptoms were such that a fatal outcome could be taken for granted. In both cases the clinical picture changed radically after the focus had been discovered in the accessory cavities and drained. After two to three weeks' treatment both patients recovered.

The pathologic changes in the nasal cavities were in the main alike in both cases, and when the maxillary sinuses were flushed, a flushing fluid was recovered which was similar in appearance in both cases. In both cases there was question not of a violent purulent and pu process but of an inflammation which sho little in the flushing fluid even when the acc sory cavities of the nose were flushed for the f time. Neither was there any swelling over, tenderness of, the palpable nasal accessory ca ties, nor was any pain present which could traced to any of the accessory cavities of nose. Thus one has here an insidious and see ingly innocent inflammation of the nasal cavi and of the accessory sinuses of the nose, a when the maxillary sinuses are flushed h scanty evidence of disease is obtained.

In both cases the lumbar punctures w carried out in conformity with the follow opinion, which I expressed in the report of m second case of acute otitis followed by strep coccic meningitis with recovery in *Acta o laryngologica* [3] and in the later article in the *Jo nal of Medical Practice* "... in a case of mening a search should be made in the first place for t seat of origin. If this is successful, the lat should be drained. Not until then should lu bar puncture be effected according to ordin indications, but with precautions to insure t least possible loss of fluid. The number of lu bar punctures should thus be as few as possibl

At the first examination I emphasized t great similarity to the first case and pronoun it as my firm opinion that this patient too wo recover if the accessory cavities of the nose w drained and lumbar punctures carried out as i dicated in the foregoing paragraph. I consi it to be absolutely necessary to employ at t same time a sulfonamide compound, and this generally known reasons. Undoubtedly, ho ever, the sulfapyridine treatment had little, any, share in the successful outcome. The p tient received relatively small quantities sulfapyridine (20.5 Gm., all told, during ele days). In the cases in which meningitis has be successfully treated with a sulfonamide pr aration, much larger quantities have, as a r been necessary. It is known from experience th the local process in the accessory cavities of t nose is practically inaccessible to peroral tre ment with a sulfonamide preparation. There no reason to suppose that it would be otherw in this case. On the other hand, there is perha reason to believe that sulfapyridine weakened t bacteria and thus contributed to the success outcome. What suggests that the sulfapyridi treatment played a minor role in this case is t fact that in the first case, which, as I have sa was almost identical with the second one, t

3. Ebert, E.: Acta oto-laryng. **19**:506, 1934.

ient recovered without being treated with fonamide drugs (which indeed were unknown that time).

Intracranial complications of infections of al cavities and accessory sinuses are rare. ly in 43 of a series of 15,000 cases was the dition due primarily to the infection of the anasal sinuses, according to Courville and senvold.[4]

It is therefore not often that an otorhinologist ounters such a complication in his practice. it is certainly still more seldom that he is led to a patient exhibiting predominant symptns of meningitis as in both these cases of ne. It is the internist who is called in and o sends such a patient to the isolation hospital the suspicion that the patient is suffering from demic cerebrospinal meningitis.

During more than fifteen years' practice as an rhinologist I have been connected with a detment of epidemic diseases for only two short iods. The first time was for three months in 29-1930; the second, for two months in 1943. ch time I have had the opportunity of attend- a patient who had been admitted with the gnosis "meningitis cerebrospinalis epidemica " with extremely serious symptoms of menitis. On both occasions it was possible to cure disease after a short time with fundamentally same therapy. It might be thought that this s due to chance. Such an attitude is, however, and in my opinion even wrong. Probably actual state of things is rather that there is orm of meningitis caused by the common pus teria which is secondary to an inflammatory cess in the accessory sinuses of the nose and ich begins with slight rhinogenous symptoms, soon forgotten or ignored by the patient and r is overlooked by the doctor. The patient ent to the isolation hospital—where as a rule re is no otologist—on suspicion of having demic cerebrospinal meningitis. He is then en to be, and is treated as, a patient with demic meningitis. When no bacteria were nd after lumbar puncture, this did not necesily change the doctor's opinion of the case. cording to Björk,[5] in the majority of cases of genic and rhinogenic meningitis a sterile lum- puncture is found. According to Neuman's at collection of statistics (quoted by Björk). terile fluid is observed to the end in about per cent of all fatal cases of otorhinogenic meningitis. Furthermore, when one finds that, according to the textbooks, epidemic cerebrospinal meningitis can be diagnosed in spite of the fact that no bacteria are reported on examination of the fluid both in direct preparations and in cultures, it is understandable that meningitis of the type I have described is incorrectly or incompletely diagnosed or otherwise overlooked. The work at both the Göteborg hospital for patients with epidemic diseases and the Boden Garrison Hospital is carried out according to the most modern principles. The otologists of the respective hospitals are consulted in every case in which meningitis is suspected to be secondary to an infection of the ears or the nose. It was thanks to this organization that the cases I have described were discovered. It is practically certain that the patients would have died if that had not been the procedure and if the paranasal cavities had not been drained as described.

How, then, is it to be explained that the repeated flushings of the maxillary sinuses and the treatment according to the method of Harke should have had such a fortunate result in both cases? In any patient, if empyema in a sinus follows a week or two of rhinitis, the empyema becomes the predominating symptom. As a rule, however, other nasal accessory cavities will probably be involved in the inflammatory process. Very likely this was what happened in these cases. It was probably from one of these other accessory cavities of the nose that the inflammatory process spread to the meninges. By flushing the maxillary sinuses (with Lichtwitz' needle) and by rinsing the nose according to the method of Harke, the other accessory cavities of the nose are also evacuated completely or partly, and this, I believe, affords an explanation of the favorable results. In a later paper I shall perhaps have an opportunity to deal in greater detail with the considerations on which this argument is based.

As already mentioned in the introduction, when my first case was published in 1931, no similar case had been previously published in the literature, nor, as far as I have been able to find out, has one been published since. Dr. A. Peyser, of Stockholm, who has, at my request, searched through all the available medical literature since 1939, reports: "There are no cases with the same etiology, therapy and recovery."

Cameron [6] published a case in which a patient with streptococcic meningitis recovered. But the morbid picture was completely different: There was a fulminant process in several of the accessory cavities of the nose, which necessitated

4. Courville, C. B., and Rosenvold, L. K.: Intranial Complications of Infections of Nasal Cavities Accessory Sinuses, Arch. Otolaryng. **27**:692 (June) 8.

5. Björk, H.: Arch. f. Hals-, Nasen- u. Ohrenh. :1, 1943.

6. Cameron, D. R.: Lancet **1**:479, 1941.

an operation, of the successful result of which the author wrote: "In the case here described, it seems unlikely that the patient would have survived without the sulphonamides."

Appelbaum [7] also mentioned a case in which a patient with streptococcic meningitis recovered; but this case does not bear any resemblance to my cases. He wote:

> . . . the meningitis was associated with a chronic sinusitis. The clinical picture was a very severe one, with marked signs of meningeal irritation, coma, and left hemiplegia. Streptococcus viridans was recovered from the spinal fluid culture. Following a course of intensive sulfanilamide therapy and spinal drainage, the patient made an uneventful recovery.

7. Appelbaum, E.: Laryngoscope **48**:482, 1938.

8. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **32**:54 (July); 256 (Aug.) 1940.

In Eagleton's [8] detailed compilation are e
merated cases in which recovery from mening
was obtained without resort to sulfonam
drugs, other factors having played a major p
in the favorable result. This work, howe
includes no rhinologic case similar to either
my 2 cases.

The same thing is true of Hagerup's co
pilation.[9] Of 1,330 patients observed in
department for diseases of the nose, ear, a
throat at the Municipal Hospital, Copenhag
Denmark, during the years from 1906 to 19
33 had intracranial complications associated w
diseases in the accessory cavities of the nose.
these, 10 survived, but not 1 of the cases
sembled mine, nor did the patients receive
same treatment.

37 Kungsportsayen.

9. Hagerup, G.: Acta oto-laryng. **24**:321, 1936.

RELATION OF THE *p*H OF NASAL SECRETIONS IN SITU TO THE ACTIVITY OF LYSOZYME

REPORT OF A CASE OF EXPERIMENTALLY INDUCED ALLERGIC RHINITIS

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In previous communications I [1] reported that the p_H of nasal secretions in situ in clinically normal nasal passages ranges from approximately 5.5 to 6.5 and that these secretions become alkaline during acute rhinitis and acute rhinosinusitis and during the more active phases of allergic rhinitis. In the light of Meyer's [2] recent accomplishment—increasing the activity of lysozyme from eight to two hundred and fifty times by the addition of a minute amount of protein—the relationship of the p_H of nasal secretions in situ to the activity of lysozyme is of interest.

Lysozyme was first described in 1922 by Sir Alexander Fleming, discoverer of penicillin. It is a substance found in various body tissues and secretions—among them, nasal secretions—which has the property of dissolving certain bacteria. Though lysozyme is most active against nonpathogenic organisms, it can attack pathogenic organisms when allowed to act in the full strength in which it occurs in some parts of the body. Fleming showed that lysozyme preparations which dissolve the susceptible organisms also kill them and inhibit their growth. It has been found that the action of lysozyme is influenced by the hydrogen ion concentration of the medium. Meyer declared that a review of the literature establishes that lysozyme activity is optimal at an acid p_H. The greatest lysis of bacterial suspensions was obtained by Boasson [3] at p_H 6.2 and by Thompson [4] at between p_H 6.05 and 6.53 (determined by glass electrode). At a point not far above neutrality an increase in the lability of lysozyme occurs, and its destruction may then ensue. Epstein and Chain [5] found that the action of lysozyme on the mucoid substrate increased with increasing acidity.

Hypersecretion of the lysozyme-secreting glands causes an eventual diminution of lysozyme in the secretions and in the secreting tissues. Hilding [6] asserted that a sharp reduction of the lysozyme content of nasal mucus, with consequently a decrease in the antiseptic properties of the nasal secretions, occurs during and preceding the invasive stages of the common cold. In some of his cases there was an increase in the lysozyme content of the secretion on the same day as the beginning of subjective improvement.

Since the action of lysozyme is known to be optimal at an acid p_H, perhaps this may explain why lysozyme is most active in the slightly acid nasal secretions of the clinically normal nose and why it is sharply diminished in the alkaline nasal secretions accompanying the acute common cold.

MEASUREMENT OF NASAL p_H

In studying the p_H of nasal secretions in situ I have employed a Coleman electrometer in conjunction with a silver–silver chloride glass electrode. The glass electrode for nasal use had been so designed and was of such proportions as to assure constant adequate contact with the mucous membranes of the lateral wall, the medial wall and the floor of the nasal cavity. The glass electrode and the tube containing a salt bridge were placed at sites approximately 1 inch (2.5 cm.) apart. The p_H readings did not differ significantly when more proximate sites were selected. Further, it was found that the variations of change with the glass electrode in situ over a period of an hour with readings taken each minute were not significantly different from the variations observed when the electrode was

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine.

1. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:150 (July) 1941; Further Studies: Significance of the p_H of Nasal Secretions in Situ, ibid. **34**:297 (Aug.) 1941; Effect of Silver Preparations and Antiseptics on the p_H of Nasal Secretions in Situ, ibid. **34**:302 (Aug.) 1941.

2. Meyer, K.: The Relationship of Lysozyme to Iodin, Science **99**:391 (May 12) 1944.

3. Boasson, E. H.: On Bacteriolysis by Lysozyme, J. Immunol. **34**:281 (April) 1938.

4. Thompson, R.: Lysozyme and Its Relation to Antibacterial Properties of Various Tissues and Secretions, Arch. Path. **30**:1096 (Nov.) 1940.

5. Epstein, L. A., and Chain, E., cited by Thompson.[4]

6. Hilding, A.: Changes in the Lysozyme Content of the Nasal Mucus During Colds, Arch. Otolaryng. **20**:28 (July) 1934.

inserted and removed every other minute over a period of one hour. An analysis was made of the variants, and reference was made to the theory of probability.

Nungester and Atkinson [7] have described a different system of measuring the p_H of nasal mucosa in situ and have reported readings in a normal group of men and women from 17 to 95 years to be from p_H 6.6 to 7.6. They found patients with acute rhinitis to have a p_H range essentially identical with that of their normal group—6.6 to 7.5— and concluded that the mean p_H value in the common cold is essentially that of the normal nasal mucosa. In an attempt to avoid possible sources of error in their procedure, a wick saturated with isotonic solution of sodium chloride was placed on the floor of the nose and connected to the calomel reference electrode by a bridge of sodium chloride.

This wick saturated with isotonic solution of sodium chloride introduced into the nose appears to explain the unusual sameness of their nasal p_H findings in health and in disease. Elsewhere I have proposed the law that the manner in which the trend of nasal p_H is affected depends on the p_H of the agent introduced into the nose. The insertion of a salt-saturated agent produces changes in the p_H of the nasal mucosa with which it comes in contact. Consequently, when readings of nasal p_H are then taken, they become in reality measurements of p_H changes produced by the saline wick rather than measurements of the true status of the nasal p_H. Such a method of procedure cannot be considered sufficiently accurate to establish a true index of the p_H of the nasal secretions in normal persons or in those with acute rhinitis.

Many investigators, including myself, maintain that under normal conditions the secretions of the nose and the sinuses maintain a biochemical composition which fluctuates within a limited range according to the demands of physiologic function. Under pathologic conditions—in infectious processes and in allergic rhinitis—certain biochemical reactions are altered. Thus, Hansel [8] found that in the acute cold the hydrogen ion concentration of the secretions and tissues in the nose is first on the alkaline side, then sharply shifts to the acid side when the stage of resolution is reached.

In the case of the p_H of nasal secretions, fluctuations in p_H appear to affect the bacterial composition. Tweedie [9] attempted to correlate the bacterial flora of the nose and the sinuses with the p_H values. A definite relationship between the two factors could be determined. It was noted that all patients with p_H values of 6.5 or below had negative cultures, although many bacteria were found in the antral smears. In addition, Tweedie made the observation that with elevation of the p_H to the alkaline side, bacteria were usually found. Similarly, Buhrmester [10] declared that all her patients with p_H 6.5 or below always had negative cultures. Both investigators expressed the belief that acidity as reflected by low values of p_H is unfavorable to the growth of pathogenic bacteria.

"In discussing changes in the p_H" Petersen wrote, "it should be emphasized that we are not dealing with marked shifts; we have to do with apparently minor alterations but which nevertheless can cause severe symptomatology. To think of the p_H of nasal secretions in terms of a static rather than a dynamic concept is to fail to realize that one is dealing with biologic structures, fluids and secretions of the human body which are constantly in motion. Thus, the pulse rate, the metabolic rate, the blood pressure, the blood count and the output of urine are never static; on the contrary, they are always dynamic expressions of activity. It would be a mistake to press all observations on patients into definite and fixed levels, quite overlooking the fact that each level can be considered only in relation to one person at a time and under certain conditions and none other.

Caution should be exercised in accepting a mean value for the p_H of nasal secretions. Nasal p_H values can no more be pegged to a single isolated "mean," such as 7, than can the normal p_H ranges of saliva (5.0 to 8.0 [12]), of the throat (4.9 to 8.0 [13]), of the skin (4.0 to 7.0 [14]) or of the midvagina (4.0 to 5.0). Each p_H range is an expression of fluctuations and variations under normal conditions and in a normal environment. As such, these p_H ranges can serve

7. Nungester, W. J., and Atkinson, A. K.: p_H of the Nasal Mucosa Measured in Situ, Arch. Otolaryng. **39**:342 (April) 1944.

8. Hansel, F. K.: Cytologic Observations on the Secretions of the Nose and Paranasal Sinuses in Allergy, Tr. Am. Laryng. A. **62**:292, 1940.

9. Tweedie, A. R.: Reaction of Nasal Mucus, Ac... oto-laryng. **24**:151, 1936.

10. Buhrmester, C. C.: A Study of the Hydrogen I... Concentration, Nitrogen Content and Viscosity of Na... Secretions, Ann. Otol., Rhin. & Laryng. **42**:1... (Dec.) 1933.

11. Petersen, W. F.: The Patient and the Weath... Ann Arbor, Mich., Edwards Brothers, Inc., 1934-19... vol. 2.

12. Grossman, L. I., and Brickman, B. M.: So... Observations on the p_H of Saliva, J. Dent. Resear... **16**:409 (Oct.) 1937.

13. Fabricant, N. D.: Introduction to the p_H of t... Throat, Arch. Otolaryng. **37**:169 (Feb.) 1943.

14. Blank, I. H.: Measurement of p_H of Skin Su... face: Technique, J. Invest. Dermat. **2**:67 (April) 19...

an expression of the dynamic concept of e secretions and tissues. The arithmetic n of arithmetic quantities is not applicable p_H which is an exponential function, for the varies not according to the actual arithmetic of the number but according to its logarithm ciprocal). In general, ordinary arithmetic ns are not applicable. Sir Napier Shaw,[15] eminent English meteorologist and mathetician, has said, "A good deal of scientific rgy has been and is being sacrificed on the r of mean values."

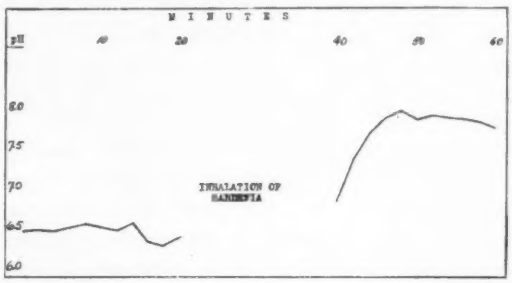
EXPERIMENTALLY INDUCED ALLERGIC RHINITIS

According to Hansel, the cytologic content of al and sinal secretions may be considered approximate index of the pathologic proces occurring in the tissues, and the cytologic ervations give important diagnostic information. He stated that in allergy it is particularly nificant that when the p_H falls low on the d side, there is a complete disappearance of eosinophils. When the p_H returns to the aline side, there is a return of the eosinophils. Polson's[16] investigations show that in allergic ients the p_H of the nasal secretions changes ard the alkaline side, and Buhrmester finds t nasal secretions from patients with nasal rgy are for the most part predominantly aline. My own studies reveal that an alkae status of the nasal secretions occurs during active stages of allergic rhinitis. The nasal descends to the neutral point and the acid tus when clinical improvement is felt by patient. Whenever the symptoms become ravated, a return to an alkaline status takes ce. Taken together, these studies indicate t physicochemical and cellular reactions play important part in allergy.

To demonstrate the dynamic ability of the al p_H to respond to an insult, an investigan of nasal p_H was undertaken in a case of perimental allergy. Admitting that allergic mptoms may develop in persons who are nsitive to flowers because of plant pollen, ederman[17] pointed out that odors emanating m flowers may also cause symptoms in susptible persons. He cited patients definitely nd sensitive to gardenias, lilacs and marids. Urbach[18] presented clinical and experimental evidence to prove that osmyls (odors) can act as allergens on inhalation in certain cases.

A young nurse known to be a sufferer from allergic rhinitis was clinically free of symptoms and findings at the time when an attack of rhinitis was experimentally induced. In the past she had been subject to frequent "colds" and intermittent attacks of asthma. Much of her difficulty was found to be due to the use of scented cosmetics; she appeared to be free from attacks of allergic rhinitis and asthma when employing unscented cosmetics. She gave a history of marked sensitivity to gardenias.

The study of the nasal p_H in this case demonstrates the dynamic ability of the nasal p_H to respond to the inhalation of the scent of a gardenia in a person known to be susceptible (figure). Readings of p_H were first taken every other minute for the purpose of determining a normal p_H range. These were then interrupted to permit the subject to inhale the odor emanating from a fresh gardenia for approximately twenty minutes. On resumption of the readings of p_H, a dramatic response occurred—the nasal p_H reached an alkalinity of p_H 7.9.



Graph showing changes of nasal p_H in a case of experimentally induced allergic rhinitis.

Strikingly enough, clinical examination at the termination of the experiment revealed a characteristic mucous membrane which one associates with an acute attack of allergic rhinitis. The subject spoke with a nasal twang and complained of nasal blockage, headache, excessive tearing and a "puffy" sensation about the eyes. A second subject who gave a questionable history of sensitivity to gardenias underwent a similar type of investigation. No changes in nasal p_H took place.

SUMMARY

The action of lysozyme is known to be optimal at an acid p_H. This may perhaps explain why lysozyme is most active in the slightly acid nasal secretions of the clinically normal nose and why it is sharply diminished in the alkaline nasal secretions accompanying the acute common cold.

A relationship of the p_H of nasal secretions to cellular reactions seems established.

The dynamic ability of nasal p_H to respond to an irritant was made evident in a case of experimentally induced allergic rhinitis.

185 North Wabash Avenue.

15. Shaw, N., cited by Petersen,[11] vol. 1, pt. 2.
16. Polson, C., in discussion on Nasal Allergy, Excluding Hay Fever, J. Laryng. & Otol. **59**:255 (June) 3.
17. Biederman, J. B.: Sensitivity to Flowers, Laryngoscope **47**:825 (Nov.) 1937.
18. Urbach, E.: Odors (Osmyls) as Allergenic ents, J. Allergy **13**:387 (May) 1942.

TREATMENT OF EXTERNAL OTITIS

II. ACTION OF SULFONAMIDE COMPOUNDS ON FUNGI ISOLATED FROM CASES OF OTOMYCOS

CAPTAIN BEN H. SENTURIA AND TECHNICIAN (5th gr.) FRED T. WOLF
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Reports from various theaters of operation indicate that external otitis is a problem of considerable importance as a cause of lost man hours of work in certain tropical and subtropical regions. There have been repeated requests for a study of this disease with a view toward devising a simple and expedient treatment which can be used under field conditions.

It is generally accepted that external otitis may be caused by bacteria, by fungi or by a mixture of bacteria and fungi. Of the varieties of fungi which have been isolated from the external ear,[1] species of Aspergillus appear to be the most frequently encountered in cases of external otitis.[2] Eleven species of the genus are listed by Dodge[3] as having been isolated from the aural canal. Penicillium and the closely related Scopulariopsis occur occasionally and are of relatively little importance.[4] The genus Mucor is represented by three species from the ear.[5] Monilia is also reported to be involved in otomycotic infections.[6] A large number of other varieties have been reported as isolated from the ear but seem to be of minor importance in comparison with those just enumerated.

The agents most frequently used in the treatment of external otitic infections at present are local applications of 95 per cent alcohol alone or of this combined with boric acid, 2 per cent salicylic acid in 70 per cent alcohol, 1 per cent thymol in alcohol, metacresyl acetate (cresatin), and potassium iodide or sulfonamide compounds by mouth. The clinical effectiveness of cresatin was emphasized by Gill[7] and has been confirm by other workers.[8] The combination of cresa with thymol has been employed with good r sults by some investigators.[9] McBurney a Searcy[10] and Schamberg and Kolmer[11] ha studied a considerable number of substances i their fungicidal and germicidal effectiveness Both in vitro and in the treatment of otomycos McBurney and Searcy found that 2 per ce thymol in alcohol was the most effective age though only slightly superior to cresatin. Alc hol, which has been widely used, was found have little fungicidal action by these workers.

A recent report[12] deals with the treatment 64 patients for external otitis, 38 of whom we treated by the local application of sulfanilamid sulfathiazole or a mixture of these with zi peroxide. The local insufflation of a mixture powdered sulfonamide compounds in cases acute external otitis resulted in a more rapi subsidence of pain and a marked decrease in th time required to effect a complete cure as com pared with other acceptable procedures. Becaus of the relative difficulty of the application cresatin and thymol by those who are n trained otologists, and since a supply of sulfo amide compounds is easily available in a theaters of operation, it was felt that the encou aging clinical results with the sulfonamide com pounds warranted further study. The prese

From the Department of Otolaryngology (Research Section), Army Air Forces School of Aviation Medicine.

1. Gill, W. D.: Otitis Externa, Ann. Otol., Rhin. & Laryng. **51**:370, 1942.

2. (*a*) Minchew, B. H.; Collins, B. E., and Harris, M. M.: External Ear Disease with Special Reference to the Fungous Type, South. M. J. **33**:1345, 1940. (*b*) Whalen, E. J.: Fungous Infections of the External Ear, J. A. M. A. **111**:502 (Aug. 6) 1938.

3. Dodge, C.: Medical Mycology, St. Louis, C. V. Mosby Company, 1935.

4. Pinkerton, M. E., and MacQuiddy, E. L.: Scopulariopsis in Otomycosis, Laryngoscope **46**:670, 1936.

5. Dowding, S., and Levey, M.: A Mould from the Ear, Canad. M. A. J. **41**:336, 1939.

6. Dobes, W. L.: Moniliasis of the External Ear Canal, South. M. J. **36**:614, 1943.

7. Gill, K.: Otitis Externa Mycotica: Comme Concerning the Prevalence, Diagnosis and Treatm of Otomycosis, Arch. Otolaryng. **16**:76 (Jan.) 1932.

8. Gill, W. D.: Mycotic Infections in Otolary ology, South. M. J. **31**:678, 1938. Johnston, W. H Otitis Externa, Ann. Otol., Rhin. & Laryng. 51 1944. Whalen.[2b]

9. Reeh, M. J.: Treatment of Otomycosis, An Otol., Rhin. & Laryng. **51**:146, 1942. Minchew a others.[2a] Whalen.[2b]

10. McBurney, R., and Searcy, H. B.: Otomycos An Investigation of Effective Fungicidal Agents f Treatment, Ann. Otol., Rhin. & Laryng. **45**:988, 1936.

11. Schamberg, J. F., and Kolmer, J. A.: Studies the Chemotherapy of Fungus Infections, Arch. Derm & Syph. **6**:746 (Dec.) 1922.

12. Senturia, B. H.: Treatment of External Otiti I. Local Sulfonamide Therapy, Laryngoscope 54:27 1944.

roject, which is concerned with the action of ndividual sulfonamide compounds on a number f fungi, was therefore undertaken.

The action of sulfonamide compounds on fungi ther than those obtained from the ear is reorded as having a bearing on the present work. linically, sulfonamide compounds have been iven by mouth in treatment for a variety of ycoses, and reports of cures by this means are n record. The oral administration of sulfonmide compounds in the successful treatment of tients for actinomycosis,[13] Madura foot,[14] torulosis,[15] blastomycosis,[16] moniliasis,[17] sporotrichosis [18] and epidermophytosis [19] has been recorded.

There has been a limited amount of previous experimentation with a view to the effects of sulfonamide compounds applied to cultures of fungi in vitro. Cutting and Gebhardt [20] studied two strains of Actinomyces hominis on mediums containing 10, 50 and 100 mg. per hundred cubic centimeters of sulfanilamide, sulfathiazole and sulfadiazine. Sulfathiazole and sulfadiazine were found to be superior to sulfanilamide in inhibiting growth of this organism. The influence of sulfon-

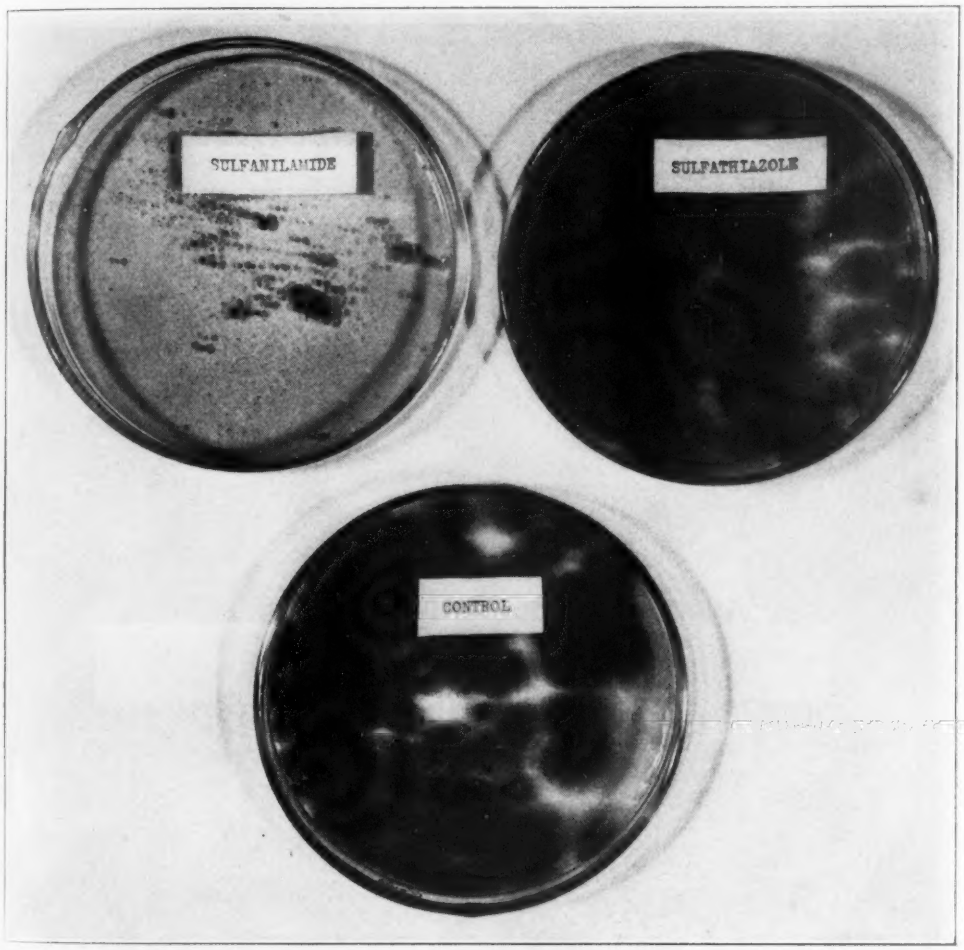

Fig. 1.—Cultures of Aspergillus fumigatus treated with sulfanilamide and with sulfathiazole showing the fungistatic action of sulfanilamide and the ineffectiveness of sulfathiazole. The control plate was untreated.

13. Walker, O.: Sulfanilamide in the Treatment of ctinomycosis, Lancet **1**:1219, 1938. Miller, E. M., and ll, E. H.: Sulfanilamide Therapy in Actinomycosis, A. M. A. **112**:731 (Feb. 25) 1939. Dorling, G. C., d Eckhoff, N. L.: Chemotherapy of Abdominal ctinomycosis, Lancet **2**:707, 1940. Ogilvie, W. H.: bdominal Actinomycosis Treated with Sulfapyridine, it. M. J. **2**:254, 1940. Dobson, L.; Holman, E., and tting, W.: Sulfanilamide in the Therapy of Actinoycosis, J. A. M. A. **116**:272 (Jan. 25) 1941.

14. Dixon, J. M.: Sulfanilamide Therapy in Madura oot, Virginia M. Monthly **68**:281, 1941.

15. Marshall, M., and Teed, R. W.: Torula Histolytica Meningoencephalitis: Recovery Following Bilateral Mastoidectomy and Sulfonamide Therapy, J. A. M. A. **120**:579 (Oct. 17) 1942.

16. Schroeder, C. B.: Ueber die Heilung eines Falles von maligner Blastomykose, Arch. f. Schiffs- u. Tropen-Hyg. **44**:477, 1940.

17. Van Bree, R. S.: Moniliasis: Sulfapyridine Treatment, J. Michigan M. Soc. **40**:197, 1941.

18. Navarro, A. M.: Sporotrichosis: Sulfanilamide Therapy, with Report of a Case, Actas dermo-sif. **32**: 271, 1940.

(Footnotes continued on next page)

amide compounds on Trichophyton has been studied independently by two groups of investigators. Lewis and Hopper,[21] working with Trichophyton gypseum, found that sulfanilamide in a concentration of 1 per cent completely inhibited growth for a period of two weeks. Dimond and Thompson [22] confirmed this observation and extended it to include Trichophyton purpureum. Other fungi, such as Monilia albicans, are completely unaffected by any sulfonamide compounds which have been tested.[21]

MATERIALS AND METHODS

Ten different isolates of fungi have been studied the course of the present work. Four of these, na Aspergillus fumigatus, Aspergillus niger, Asperg glaucus and Aspergillus sydowi, were isolated from external auditory canals of patients exhibiting symp of otomycosis. The remaining strains, one of A. ni one of Penicillium spinulosum, three of M corymbifer and one of Monilia albicans, were isol from sources other than ears but belong to sp known to cause otomycosis.

The fungi were grown on Sabouraud's agar. cultures were grown in an incubator in which a cons

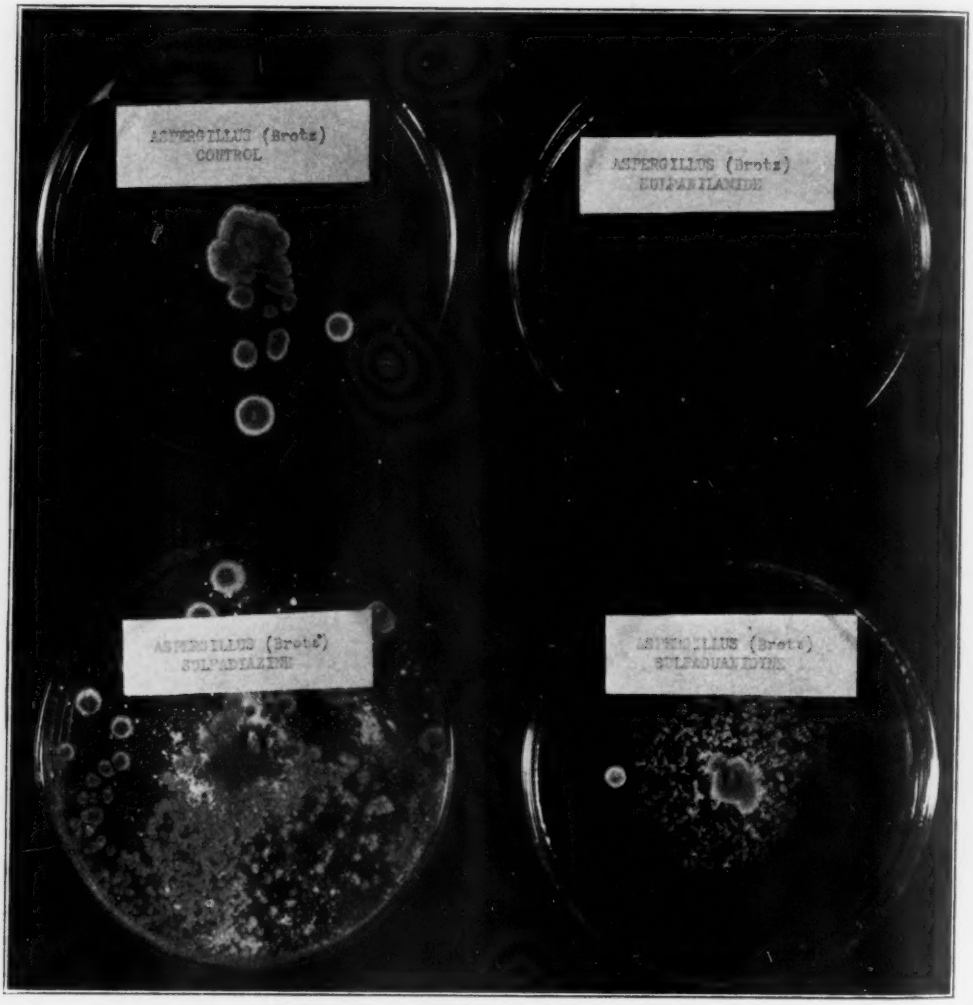

Fig. 2.—Cultures of Aspergillus sydowi showing the action of sulfanilamide, sulfadiazine and sulfaguanidine comparison with an untreated control plate.

19. Pfalzgraf, C.: Prontosil-Alcohol-Acetone Solution in Therapy of Epidermophytosis and Moist Eczema, München. med. Wchnschr. **88**:399, 1941.

20. Cutting, W. C., and Gebhardt, L. P.: Inhibitory Effects of Sulfonamides on Cultures of Actinomyces Hominis, Science **94**:568, 1941.

21. Lewis, G. M., and Hopper, M. E.: Effect of Sulfanilamide and Its Derivatives on Fungi, Arch. Dermat. & Syph. **44**:1101 (Dec.) 1941.

temperature of 37 C. was maintained. The sulfona compounds used were sulfanilamide, sulfathiazole, s diazine, sulfaguanidine and sulfamerazine. These c pounds were not incorporated into the medium as been done by previous workers,[23] but were powde

22. Dimond, N. S., and Thompson, K. W.: Effect of Sulfonamide Drugs on Trichophytons in V J. Invest. Dermat. **5**:397, 1942.

23. Cutting and Gebhardt.[20] Lewis and Hopp Dimond and Thompson.[22]

r the surfaces of the plates immediately after inocula-
s in an attempt to duplicate as closely as possible
method described [12] in the treatment of infected
itory canals.

EXPERIMENTAL RESULTS

In at least some cases of otomycosis it may be
erved that the fungus appears to grow and
rulate in the cerumen within the external
ditory canal. Therefore, it was of interest to
ermine whether these fungi from the ear
ld grow in vitro on a medium containing
cerumen-agar were inoculated with a culture of A. fumigatus from an aural canal. Other similar plates were inoculated with a strain of A. niger. On this medium both A. fumigatus and A. niger grew and sporulated within three to five days. No growth of either organism was observed on control plates containing pure 2 per cent agar but no cerumen. A liquid medium was also prepared containing only sterile cerumen and distilled water. Growth of A. fumigatus was first evident after three days, but a period of nine days was

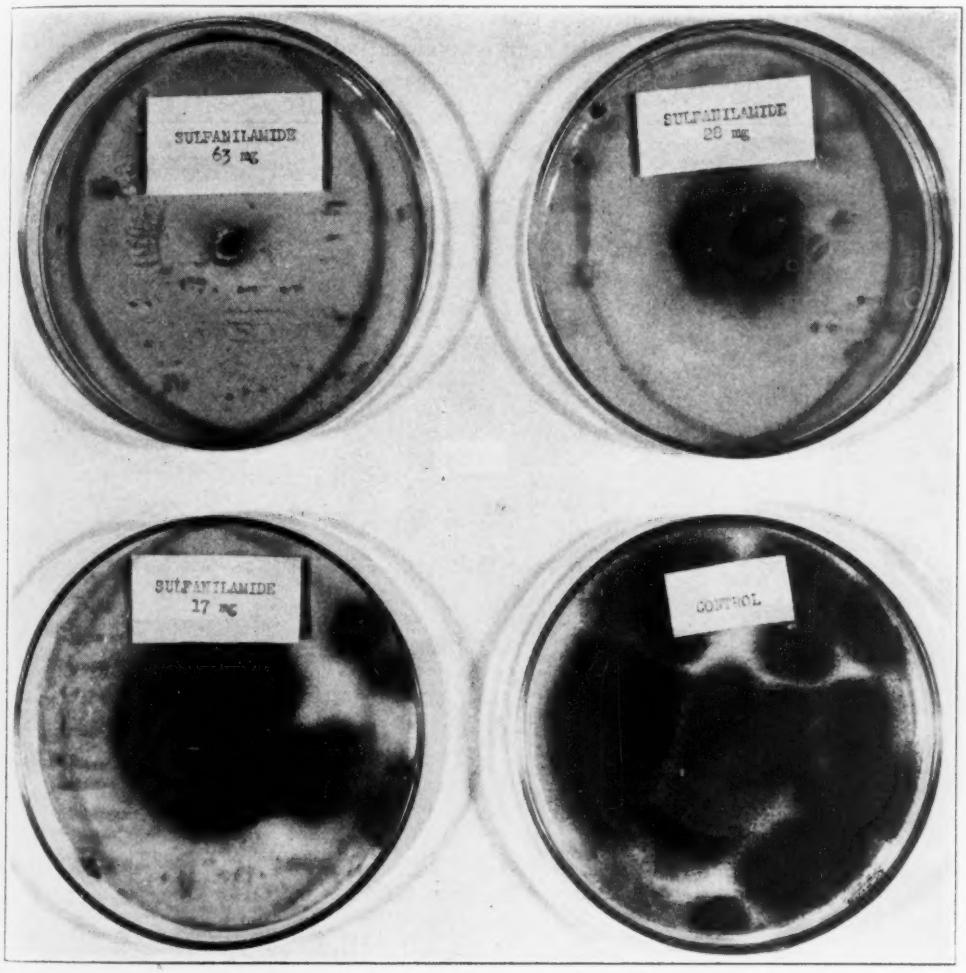

Fig. 3.—Cultures of Aspergillus fumigatus showing the fungistatic influence of 63, 28 and 17 mg. of sulfanilide insufflated on the surface of the medium. The control plate was inoculated at the same time but received no sulfanilamide.

rumen as the sole nutrient material. Dowding
d Levey,[5] working with Mucor corymbifer and
ucor circinelloides, succeeded in cultivating
ese organisms on sterilized cerumen, but no
ilar experiments with species of Aspergillus
e ever been made.

A medium containing 1 per cent cerumen,
per cent agar and distilled water was therefore
pared and sterilized by autoclaving. Plates of
required for the formation of a mat of mycelium and the production of abundant spores on the surface of the medium.

In a previous report [12] it was stated that a mixture of sulfanilamide, sulfathiazole and zinc peroxide (4:2:2) was effective against acute otomycosis. An experiment was therefore designed to test the effect of this mixture on cultures of fungi in vitro. Plates of Sabouraud's

agar were inoculated with A. fumigatus, one group being dusted with varying amounts of the mixture and another group being left untreated to serve as a control. In several experiments of this type the diameter of the colonies of the fungus on the treated plates did not exceed 1 cm. after a five day period of growth, while control plates were completely covered by sporulating mycelium at this time. It is thus apparent that this mixture of sulfonamide compounds has a pronounced inhibitory effect on the growth of this organism.

In order to test the separate components of the mixture, duplicate plates of Sabouraud's agar were inoculated with A. fumigatus and treated as follows: One group of plates was dusted with sulfanilamide alone; one group was dusted with sulfathiazole alone; one group was dusted with a mixture of sulfanilamide, sulfathiazole and zinc peroxide; one group was left untreated. The control plates and the plates treated with sulfathiazole were completely covered by mycelia and spores of the fungus within a few days, whereas plates treated either with sulfanilamide or with the mixture of sulfonamide compounds showed almost complete inhibition of growth of the fungus (figs. 1 and 2). It is thus apparent that the fungistatic effect of the mixture of sulfonamide compounds is due to the sulfanilamide it contains. Sulfathiazole appeared to almost completely ineffective in inhibiting gro of A. fumigatus.

In order to determine the possible influe of zinc peroxide on the action of sulfanilam quantitative experiments were undertaken which one series of cultures was treated w known amounts of sulfanilamide and a simi series of cultures was treated with kno amounts of sulfanilamide plus zinc perox (4:1). A comparison of the amou of growth in the two series of plates, the basis of equal amounts of sulfanilam applied, indicates that zinc peroxide does increase the effectiveness of sulfanilamide aga

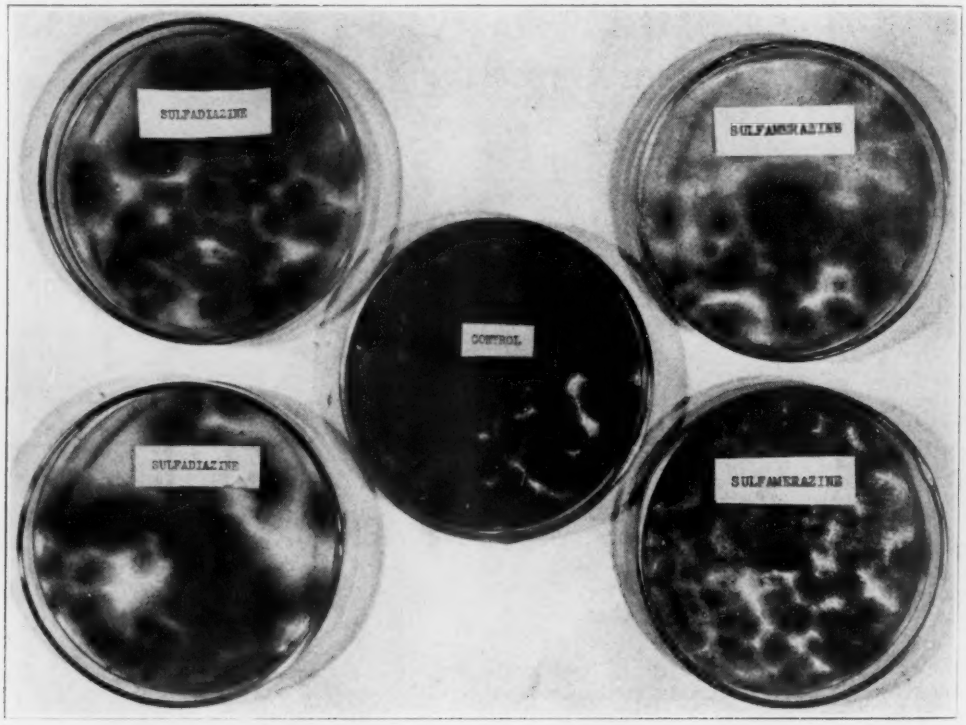

Fig. 4.—Cultures of Aspergillus fumigatus treated with sulfadiazine and sulfamerazine showing the rela ineffective fungistatic action of these compounds. The control plate was untreated.

fungi. However, zinc peroxide may be utili for its effect against anaerobic and microae philic organisms [24] without interfering with t fungistatic action of sulfanilamide.

Experiments were conducted in which kno amounts of powdered sulfanilamide were appli to cultures of fungi. In one such quantitati experiment, six plates of Sabouraud's agar i oculated with A. fumigatus were treated wi powdered sulfanilamide distributed evenly ov

24. Johnson, B. A., and Meleney, F. L.: The An septic and Detoxifying Action of Zinc Peroxide Certain Surgical Aerobic, Anaerobic and Micro-Aer philic Bacteria, Ann. Surg. **109**:881, 1939.

surface of the plates from an insufflator. In order to determine the amounts of sulfanilamide applied, the insufflator was weighed before and after each application. The doses varied from 1 to 63 mg. per plate. Growth of the fungi was found to be in inverse proportion to the dose used (fig. 3). Quantities above 20 to 30 mg. of sulfanilamide per plate resulted in practically complete inhibition of growth of the fungi for a period as long as two weeks. In treatment for acute otomycosis in the typical over each plate. No growth occurred on the plates dusted with sulfanilamide, while multiple colonies appeared on those dusted with sulfathiazole and on the untreated control plates.

In addition to sulfanilamide and sulfathiazole, three other sulfonamide compounds, namely, sulfadiazine, sulfamerazine and sulfaguanidine, were tested. None of these had any observable effect on the growth of A. fumigatus (fig. 4). It was believed that the results of these experiments, in which only sulfanilamide was shown

Fig. 5.—Cultures of Mucor corymbifer contrasting the action of sulfanilamide, sulfadiazine, sulfathiazole and sulfaguanidine. The control plate was untreated.

clinical case the amount of sulfanilamide applied to a single ear will be in the order of 100 mg., thus far exceeding the amount required to check the growth of the organism under cultural conditions.

In order to determine the possible prophylactic value of sulfonamide compounds, a series of plates was dusted with sulfanilamide and sulfathiazole. One loopful of spores of A. fumigatus was shaken to be effective, might be influenced by the physical state of the compound which was applied to the cultures. Therefore, experiments were conducted to compare the results obtained with the various compounds in powdered, tablet and crystalline form. The tablets, which presumably contained certain binding substances in addition to the sulfonamide compound, were ground up with a mortar and pestle before being applied

to the plates. The results of the experiments indicated that the crystalline and tablet forms of sulfanilamide are as effective as the powdered product.

The preceding experiments have dealt almost entirely with A. fumigatus. Tests of the effect of various sulfonamide compounds on A. niger, A. glaucus, A. sydowi and Penicillium spinulosum were accordingly performed in the manner already described for A. fumigatus. All of these organisms were inhibited by sulfanilamide and organism (fig. 5). Monilia albicans (fig. was the only organism tested which was affected by sulfanilamide. This result confir the finding of Lewis and Hopper.[21]

In summary, the results of these experime with a number of available sulfonamide co pounds show that only sulfanilamide is effect in inhibiting the growth of the fungi which h been tested. It may be concluded that in ca of external otitis in which Aspergillus, Peni lium or Mucor may be found, the local appli

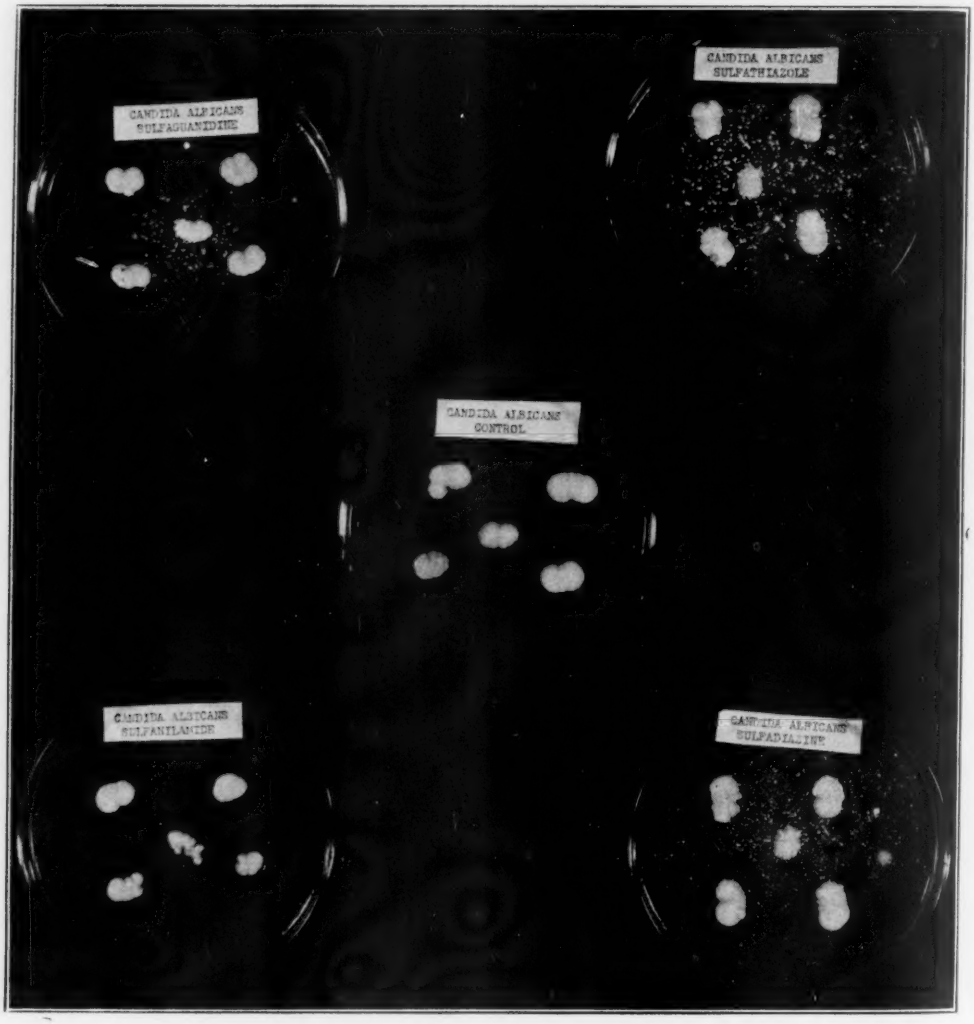

Fig. 6.—Cultures of Monilia (candida) albicans showing the absence of any fungistatic effect of sulfanila sulfathiazole, sulfadiazine or sulfaguanidine.

not by other sulfonamide compounds, just as in the case of A. fumigatus. The influence of various sulfonamide compounds on three strains of Mucor corymbifer was also studied. The cultures treated with sulfanilamide showed pronounced fungistasis, although the inhibition of growth of Mucor was not as great as with the various species of Aspergillus. Other sulfonamide compounds are ineffective against this tion of sulfanilamide should be accompanied beneficial results. No fungistatic effect sh be expected where Monilia is concerned.

CONCLUSIONS

1. Sulfanilamide has a marked fungistatic ef when applied to the surface of cultures of Asp gillus, Penicillium and Mucor.

2. Monilia is unaffected by sulfanilamide.

3. Sulfathiazole, sulfadiazine, sulfaguanidine and sulfamerazine have no observable effect on the growth of these fungi.
4. Cerumen will support the growth of fungi.
5. The local application of powdered sulfanilamide in cases of otomycosis, with the exception of those caused by Monilia, should be accompanied by beneficial results.
6. The use of sulfanilamide as a prophylactic against recurring otomycotic infections should receive further study.

Dr. N. F. Conant, of the Duke University School of Medicine, Dr. Victor M. Cutter Jr., of the department of botany of Cornell University and Dr. F. A. Wolf, of the department of botany of Duke University, provided or identified some of the cultures of fungi used in this study; Miss Beatrice Stevens, of the Army Air Force School of Aviation Medicine, assisted with the laboratory work, and Mr. John J. Tucker, chief of the laboratory of photography of the Army Air Force School of Aviation Medicine, aided with photography.

CAROTID BODY TUMOR OF THE MIDDLE EAR AND MASTOID

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Precise knowledge of the structure of the carotid body dates back to the contribution of von Luschka [1] in 1862. Since the communications of Marchand,[2] von Paltauf,[3] Mönckeberg [4] and others dealing with cases of tumor of the carotid body, numerous contributions to the anatomy, embryology, physiology and pathology of the carotid body have been made.

The literature dealing with tumor of the carotid body has been thoroughly reviewed by Keen and Funke [5] (1906), Callison and Mackenty [6] (1913), Bevan and McCarthy [7] (1929), Rankin and Wellbrock [8] (1931), Peterson and Meeker [9] (1936), Sonck [10] (1938), Gratiot [11] (1943) and others. From these collective reviews it appears that to date there have been approximately 300 cases of tumor of the carotid body, an indication that tumors of this t must be regarded as rare.

Even less frequent, however, are the repo of cases of aberrant carotid body tumor. Ri ing [12] reported a case in which at postmort examination an aberrant carotid body tumor discovered entirely apart from the norma situated carotid body. Traina [13] describe histologically proved carotid body tumor loca at the angle of the jaw, below the mastoid pr ess, which was clinically assumed to be a d moid cyst. Clagg [14] reported a case in wh the tumor occurred in the usual site, in neck. At autopsy, the patient was found have two histologically identical carotid b tumors involving the Zuckerkandl bodies in abdomen. Goodof and Lischer [15] reported case in which a carotid body tumor was d covered in the pancreas at autopsy.

The occurrence of a carotid body tumor the middle ear and extending into the mast process in the absence of a demonstrable tu of the carotid body in the neck, however, i not hitherto been described, and because of extreme rarity of this condition a descript of this case is warranted.

REPORT OF A CASE

History.—M. R., aged 36, was admitted to the o logic service of Mount Sinai Hospital, complaining a "growth" in the external canal of the left ear a of deafness. Except for the fact that he had alw been somewhat retarded mentally, the past history w without bearing on the case. The patient declared had been almost completely deaf in his left ear for least ten years. He did not recall the precise on of the deafness, nor was he aware of any signific progression. He disclaimed all pain or discharge in the left ear at any time.

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1. von Luschka, H.: Ueber de drüsenartige Natur des sogenannten Ganglion intercaroticum, Arch. f. Anat. u. Physiol., 1862, pp. 405-414.

2. Marchand, F.: Beiträge zur Kenntniss der normalen und pathologischen Anatomie der Glandula carotica und der Nebennieren, in Festschrift Rudolph Virchow, Berlin, G. Reimer, 1891, vol. 1, p. 535.

3. von Paltauf, R.: Ueber Geschwülste der Glandula carotica nebst einen Beitrage zur Histologie und Entwickelungsgeshichte derselben, Beitr. z. path. Anat. u. z. allg. Path. **11**:260-301, 1891-1892.

4. Mönckeberg, J. G.: Die Tumoren der Glandula carotica, Beitr. z. path. Anat. u. z. allg. Path. **38**:1-64, 1905.

5. Keen, W. W., and Funke, J.: Tumors of the Carotid Gland, J. A. M. A. **47**:469-479 (Aug. 18); 566-570 (Aug. 25) 1906.

6. Callison, J. G., and Mackenty, J. E.: Tumors of the Carotid Body, Ann. Surg. **58**:740-765, 1913.

7. Bevan, A. D., and McCarthy, E. R.: Tumors of the Carotid Body, Surg., Gynec. & Obst. **49**:764-779, 1929.

8. Rankin, F. W., and Wellbrock, W. L. A.: Tumors of the Carotid Body, Ann. Surg. **93**:801-810, 1931.

9. Peterson, E. W., and Meeker, L. H.: Tumors of Carotid Gland, Ann. Surg. **103**:554-571, 1936.

10. Sonck, C. E.: Surgical Therapy of Tumors of Carotid Body as Observed in Finland, Internat. Abstr. Surg. **66**:518, 1938; in Surg., Gynec. & Obst., June 1938.

11. Gratiot, J. H.: Carotid Body Tumors: Collective Review, Internat. Abstr. Surg. **77**:177-186, 1943; in Surg., Gynec. & Obst., September 1943.

12. Rössing, F.: Struma eines akzessorischen Glo caroticum, Arch. f. klin. Chir. **181**:571-574, 1935.

13. Traina, S.: Submastoid Tumor Developed fr Aberrant Germ of Carotid Gland and Clinically Simu ing a Dermoid Cyst, Valsalva **8**:450-454, 1932.

14. Clagg, R. W.: Concurrent Tumors of Left Car Body and Both Zuckerkandl Bodies, Arch. Path. 635-645 (Nov.) 1934.

15. Goodof, I. I., and Lischer, C. E.: Tumor of Carotid Body and Pancreas, Arch. Path. **35**:906 (June) 1943.

Seven weeks before admission to the hospital, while being examined by his local selective service board, he was informed that he had a "bad left ear." He consulted a specialist, who removed some tissue from the canal in the external canal for a biopsy. According to the physician, the removal of tissue was associated with profuse bleeding, and the mass was reported as "vascular granulation tissue."

Four weeks prior to admission to Mount Sinai Hospital the intermittent purulent discharge from the left ear was noted, and one week later the patient was aware of facial weakness on the left side. He had no headache, dizziness, tinnitus or diplopia at any time.

Examination.—This young man was well developed and well nourished and did not appear ill. He had peripheral facial weakness on the left side. The chorda tympani nerve was not involved. The right drum membrane had a few small calcified plaques in its anterior portion but was otherwise normal. The landmarks were present. In the left ear a large reddish mass, simulating granuloma, completely filled the depths of the external auditory canal. There was present a scant thick creamy discharge, nonpulsating and nonfetid. The fistula test was negative. The patient could not hear a loud spoken voice with the Bárány noise apparatus in the right ear. There was left peripheral facial palsy. The fundi oculi were normal. The larynx was normal. Neurologic examination disclosed no abnormality.

The hemoglobin content was 89 per cent. The white blood cell count was 7,400, with segmented polymorphonuclears 67 per cent, nonsegmented polymorphonuclears 4 per cent, lymphocytes 22 per cent, monocytes 7 per cent and eosinophils 4 per cent. The Wassermann and Kahn tests of the blood were negative. The blood pressure was 140 systolic and 90 diastolic. The pulse rate was 90. Urinalysis gave entirely negative results.

Roentgen examination of the mastoid processes and the petrous pyramids revealed good pneumatization on both the right and the left side. The intercellular septums on the right side were intact and showed no evidence of destruction. On the left side there were marked clouding of the periantral region and decalcification of the septums in the perisinal region. There was no distinct abnormality in the petrous bone.

Determinations of chronaxia showed a reaction of degeneration in the involved left facial nerve. Repeated determinations throughout the patient's stay in the hospital showed little significant change.

Operation.—On April 18, 1942 an exploratory radical mastoidectomy was performed on the left side. There was excessive bleeding from the bone, which showed widespread softening, but there was no frank pus in the mastoid cavity. In the initial groove near the tip the disease process had exposed the facial nerve, which was embedded in what appeared to be vascular granulation tissue. The bony canal wall was abnormally friable. When it was taken down, the middle ear was found filled with a large purplish mass, which appeared continuous with the exposure in the initial groove and the posterior canal wall near the tip.

It was possible to delimit the tumor anteriorly, superiorly and medially, but not inferiorly, where it appeared firmly attached in the hypotympanum and in the region of the stapes and the fallopian canal. There was no fistula of the horizontal canal. The tumor did not pulsate; it was fleshy, encapsulated and without bruit.

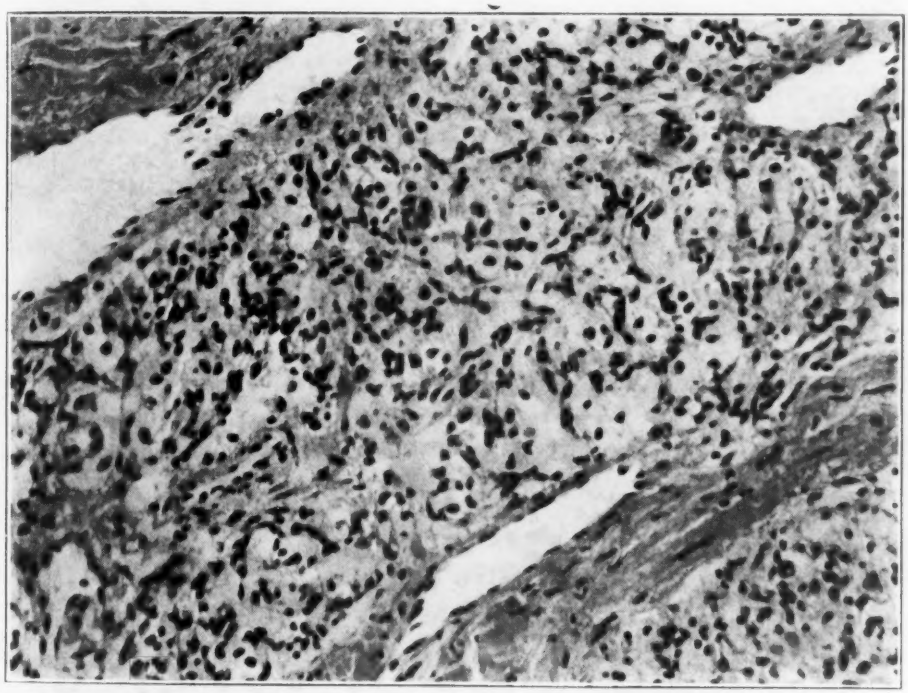

Fig. 1.—A representative area of the tumor from the tympanic cavity. Note the characteristic tumor cells in groups separated by dense fibrous tissue stroma. The empty spaces indicate the vascular nature of the tumor. ×75.

In view of the extent of the tumor and the profuse bleeding on manipulation, I felt that I was dealing with a vascular neoplasm, possibly a hemangioma. On probing the floor of the middle ear, I could feel no bone. The possibility that the broad firm attachment in the hypotympanum might indicate involvement of the jugular dome was also considered. The tumor mass was excised, only the hypotympanic attachment being left in situ. The radical mastoidectomy was then completed. The wound was left open, and the cr was loosely packed with iodoform gauze. A P plastic flap operation was not performed, as it was that this might be better done at a later date, par larly since the exact nature of the pathologic pro was not then known

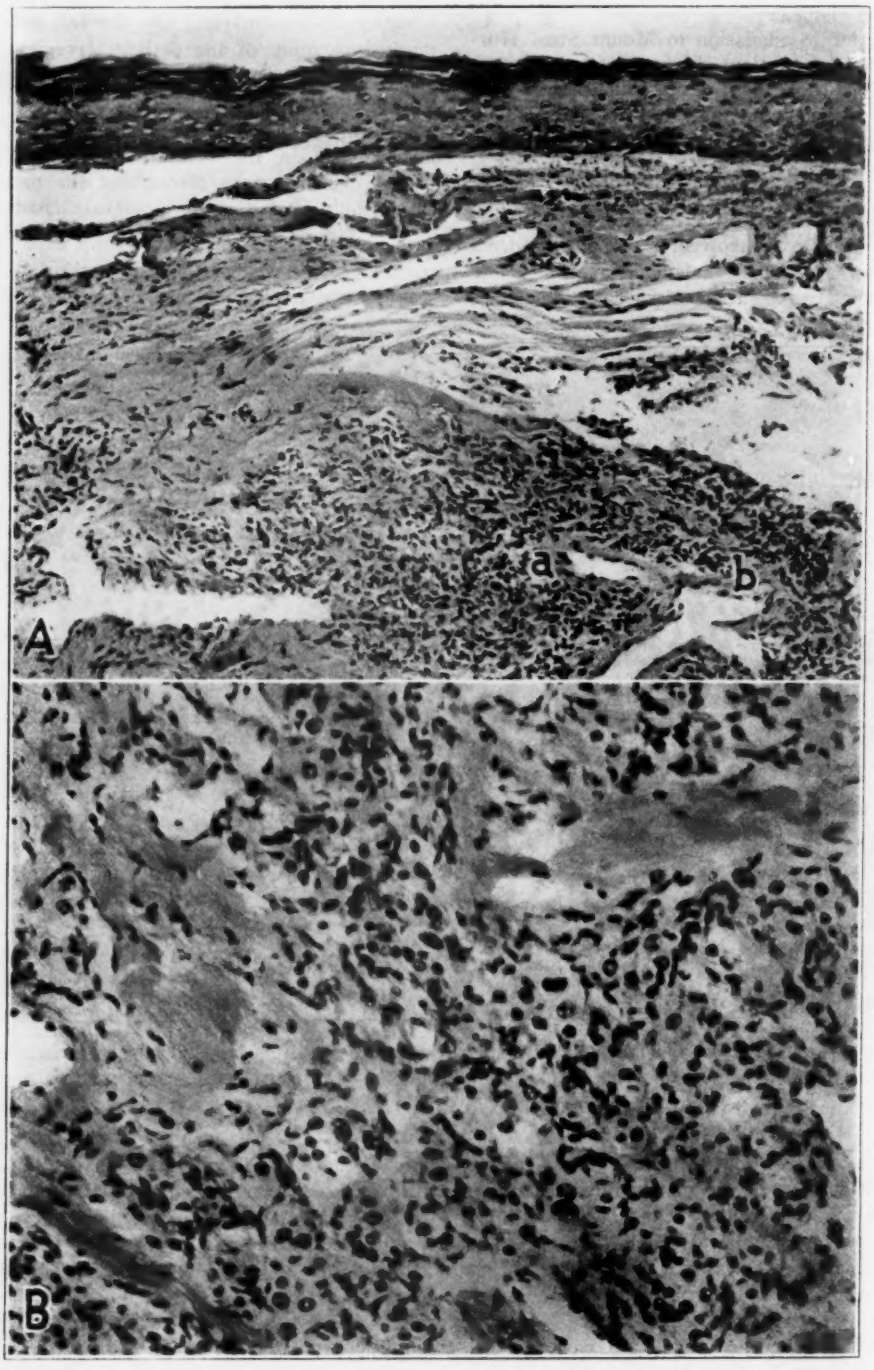

Fig. 2.—*A*, low power photomicrograph of a biopsy specimen from the mass in the external canal. Cell areas represent the tumor (*a*) and empty spaces vascularity of the tumor (*b*). × 115. *B*, photomicrograph ta from a typical carotid body tumor in the same magnification as that shown in figure 1. Note the identical hi logic features of this tumor and the tumor in figure 1. × 275.

The postoperative course was uneventful. There no change in the facial paralysis. Because of the op tive findings, I urged that the patient should be tre

hough the tumor were a malignant one, particularly examination of the excised tumor revealed puz- pathologic features. While waiting for a definite logic report, the patient received intensive radia- therapy.

thologic Report.—Pathologic examination revealed unusual features for a tumor in this location. Sadao Otani reported as follows:

he fragment of tissue (measuring approximately 7 mm.) is partially covered by a squamous cell . Immediately beneath this layer is an unusual r structure. Large tumor cells in groups are rated by dense fibrous tissue septums, which con- dilated veins. The groups of tumor cells are ered by capillaries and form small alveolar struc- . This alveolar arrangement of the neoplastic however, is not always distinct; in places the cells the form of compressed cordlike structures. The preserved tumor cells are large and polyhedral; nuclei are rather small and not hyperchromatic. large cytoplasm is frequently vacuolated. This olation obscures the cell outline. The oval nuclei uniform, although there are areas in which the r cell nuclei vary in shape. No mitoses were found 1). The excised specimen was received in a solu- of formaldehyde, and chromaffine pigment of the r cells could therefore not be demonstrated. The al histologic picture, however, is that of carotid tumor."

previous biopsy specimen removed from the mass e aural canal by Dr. A. Moshinsky presents a re identical with that in my specimen except, per- for an absence of clear alveolar structures 2*A*). Dr. Otani gave me permission for the use gure 2*B*, a section made from a proved carotid tumor of the neck. The structural identity evi- on comparing figures 1 and 2*A* and *B* is striking definitely seems to substantiate the diagnosis of a id body tumor.

e patient was last seen in the follow-up clinic on h 26, 1944, about two years after operation, at h time he looked well and had gained weight. facial weakness was unchanged. The external ory canal was narrowed and dry. There was no ation or bleeding on wiping the narrowed canal. e was no evidence of recurrence. The postauric- wound was healed. There was no enlargement ands or difficulty in swallowing. The neurologic s was normal. The throat, the neck and the x were entirely normal.

COMMENT

ccording to Goodof and Lischer,[15] in all reported cases of tumor of the carotid body, tumor has been localized in the neck, never ading beyond the lymph nodes in the im- iate neighborhood of the primary mass. y further stated that the cases reported by ord and Davis and Mönckeberg are the ones on record in which autopsy disclosed mor outside the neck. Goodof and Lischer ribed what they believed to be the first nce of a true carotid body tumor discov- in the pancreas, in a patient with a tumor e carotid body in the neck in the usual site.

In 1911 Matthews[16] reported before the New York Surgical Society a case in which he removed part of the adherent tumor. Two years later the patient was operated on by Dr. D. MacPherson at the Post-Graduate Medical School and Hospital for a tumor presenting in the external canal, on the same side as the tumor of the neck. Biopsy was attended with profuse bleeding. Unfortunately, the biopsy specimen was lost. This case must be regarded as demonstrating prolongation upward of the growing tumor in the neck and not a primary tumor in the ear.

Of extreme interest and possibly providing, theoretically at least, a morphologic explanation of the presence of a carotid body tumor in the middle ear and the mastoid process is a communication of Dr. Stacy R. Guild,[17] which states:

> Human temporal bone sections reveal structures in several respects like the carotid body for which the name glomus jugularis is proposed. Usually they are in the adventitia of the dome of the jugular bulb, immediately below the bony floor of the middle ear and near the ramus tympanicus of the glossopharyngeal nerve. Usually there is but a single flattened ovoid glomus, about 0.5 mm. in the longer diameters and about 0.25 mm. thick. Occasionally two or more smaller bodies are present; sometimes one or all are in the canal that transmits the ramus tympanicus through the floor of the middle ear (in one case also along the course of this nerve over the cochlear promontory).
>
> Each glomus, wherever located, consists of blood vessels of capillary or precapillary caliber with numerous epithelioid cells between the vessels. . . . Innervation and blood come from the same trunks that supply the carotid body; namely, glossopharyngeal nerve and ascending pharyngeal artery (through its inferior tympanic branch).

The possibility that the tumor herein reported may have developed from the glomus jugularis, first named by Guild, is tentatively proposed.

The complaint of deafness in the left ear of ten years' duration, if explained on the basis of the carotid body tumor, is in keeping with the known slow growth of such tumors, particularly since a discharge from the ear was never noted.

SUMMARY

The carotid body tumor reported involved the middle ear and the mastoid bone and occurred without any clinically demonstrable tumor in the usual site in the neck.

101 East Seventy-Third Street.

16. Matthews, F. S.: Carotid Body Tumor: Report of Case, Ann. Surg. **59**:752-753, 1913.

17. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis, in Man, Anat. Rec. (supp. 2) **79**:28, 1941.

DETERMINATIONS OF INTRANASAL *p*H

A DISCUSSION AND CRITICISM

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Confusion is evident in such experiments as have been reported for the determination of intranasal p_H. The cause seems to lie chiefly in unawareness that within the nasal chambers are two distinct sources of hydrogen ion concentration: living tissue and nonliving secretion. The purpose of this discussion is to suggest a more valid premise for such experimentation. It is hoped that with this premise more accurate and therefore more useful conclusions may be drawn.

In the opening paragraphs of his "Introduction to Comparative Biochemistry" Baldwin [1] stated: "In the body of the animal the properties of the perfusing fluid are kept remarkably constant. The total salinity, the p_H and the temperature remain constant within very small limits. A large part of physiology is concerned with the mechanism whereby this constancy is maintained."

The term "body fluid" embraces both cellular and extracellular fluids, which together comprise the liquid content of the tissues. While differing significantly in chemical constituents, cellular and extracellular fluids maintain identical physical characteristics. This subject is magnificently treated by Gamble [2] in a paper that should be perused by all who contemplate experimentation with tissue fluids or their derivatives. Approximate figures for the physical properties of body fluid are generally known and agreed on and require no elaboration.

Nasal mucous membrane, being living tissue, is perfused with body fluid just as are all other living tissues, and there is no reason to doubt that this fluid maintains standard physical values.

Not included in the category of body fluid are secretions and excretions on the body surfaces. While derived from body fluid, these products are nonliving, and their physical properties are neither standard nor constant, because they no longer are under the control of body mechanisms. Nasal secretion, for example, regardless of its properties at the time of ori maintains no physical standards. Released f "the mechanism whereby constancy is m tained," it is modified by a multitude of envi mental factors. Its temperature, hydrogen concentration and tonicity change literally every breath. The assumption that cha occur in it only after it is removed from nasal fossae is erroneous. Changes occur f the moment of production.

Thus there are in contiguity two intran fluids: body fluid within the tissue with phys characteristics controlled and constant, secretion on the tissue surface with values controlled and ever changing.

Experimental determinations of the phys properties of nasal fluids have been reported many years. Usually interest has been in hydrogen ion concentration of nasal secret sometimes in that of secretion in situ, so times in that of secretion removed. Determi tions on secretion in situ generally have sho a narrower range of variation than determi tions on secretion removed from the nose.[3]

Separate determinations of the hydrogen concentration of either nasal secretion or m mucous membrane in situ seem to be impr ticable by present methods. The term "intran p_H" would seem to define more accurately s values as have been reported. Secretion membrane are so intimately associated that indicator placed intranasally is certain to cont both, to respond to both and therefore to g a combined value. However, experimental ports suggest that the values may be weigh in favor of one or the other. Nungester Atkinson [4] observed that "good" contact of el trode with mucous membrane gave constant readings only slightly below the standard body fluid but that "incomplete" contact of el trode with membrane gave p_H readings lo

1. Baldwin, E.: An Introduction to Comparative Biochemistry, London, Cambridge University Press, 1937.

2. Gamble, J. L.: Extracellular Body Fluid, Internat. Clin. **2**:184 (June) 1936.

3. Tweedie, A. R.: Nasal Flora and Reaction Nasal Mucus, J. Laryng. & Otol. **49**:586 (Sept.) 1

4. Nungester, W. J., and Atkinson, A. K.: *p* the Nasal Mucosa Measured in Situ, Arch. Otolary **39**:342 (April) 1944.

less constant. This suggests that firm contact gives a proportionate increase of membrane in the combined value, whereas loose contact reduces this proportion and gives a value more nearly that of secretion. But this is not an explanation offered by those workers. They make no statement that would indicate awareness of nasal secretion and nasal mucous membrane as separate sources of hydrogen ion concentration.

Fabricant,[5] employing an intranasal electrode, made determinations which he reported as the p_H of nasal secretion in situ. He made no statement that would indicate awareness of nasal membrane and nasal secretion as separate sources of hydrogen ion concentration. Yet his electrode could not have avoided contact with both sources and could give only combined values. His reported findings are considerably below the standard for body fluid and suggest what Nungester and Atkinson referred to as incomplete contact of electrode with membrane. Convinced of the importance of his findings, Fabricant announced that they indicated a purposeful acid barrier against infection, and both his findings and his conclusions were widely accepted without confirmation or critical analysis.

Nungester and Atkinson,[4] employing an intranasal electrode, made determinations of what they reported as the p_H of nasal mucous membrane. Their method was similar to Fabricant's with corrections to insure greater accuracy. They concluded that their determinations of p_H indicate a neutral or slightly alkaline state "in normal persons as well as in those with acute rhinitis." They explained Fabricant's low p_H values as due to faulty technic. The reader is justifiably astonished at what amounts to interchangeable usage by these workers of the words "mucus" and "mucosa." This interchange is maintained throughout their report and appears to have important bearing on their conclusions. They referred to Fabricant's determinations as nasal, whereas that worker reported his findings as from nasal secretion. They wrote that they "reinvestigated the p_H of nasal secretions measured in situ" but described only what they stated were values from the mucous membrane. In contrasting their findings with Fabricant's they explained the differences as due to technic. They seem to have been completely unaware that Fabricant reported his values as from nasal secretion whereas theirs were reported as from nasal mucous membrane.

In view of the foregoing considerations it seems appropriate to say that in experimental determinations of intranasal p_H certain conditions so far unrecognized must be accounted for in order to validate both premise and conclusion. An intranasal electrode contacts both nasal secretion and nasal mucous membrane. Both are sources of hydrogen ion concentration, the secretion being uncontrolled and inconstant and the membrane being controlled and constant. Misleading, therefore, are reports of in situ determinations of p_H as being specifically of nasal secretion or of nasal mucous membrane wherein these conditions have not been accounted for. More correctly, these determinations simply are those of intranasal p_H.

In a previous paper[6] I discussed some of the factors that change the physical characteristics of nasal secretion in situ. A brief comment seems advisable.

The temperature of nasal secretion is changed in situ by nasally respired air through contact and evaporation. This seems sufficiently obvious to need no elaboration. The change of the temperature usually is downward.

The osmotic pressure of nasal secretion in situ is changed by nasally respired air through evaporation and concentration. This effect is modified by the degree of nasal ventilation and by the rate of production of secretion. The modification of the osmotic pressure usually is toward hypertonicity.

The hydrogen ion concentration of nasal secretion is changed in situ by two main factors having opposite effects. Absorption of carbon dioxide from nasally respired air causes increase of hydrogen ions with decrease of p_H. Proteolysis results in combining of hydrogen ions with protein split products, causing increase of p_H. These effects are modified by the degree of nasal ventilation and by the viscosity and the composition of nasal secretion, the rate of its production and its proximity to the nasal air stream. The modification of p_H usually is toward acidity, but with much discharge and blockage it may be toward alkalinity.

It is apparent that changes in the physical properties of nasal secretion in situ are logical and to some extent predictable. It cannot be said that they are to be interpreted as necessarily purposeful. They indicate, if anything, the tolerance of the nasal membrane to changes in external environment.

Intranasal treatment is treatment of mucous membrane; it is not treatment of nasal secretion. Therefore, in designing solutions for nasal

5. Fabricant, N. D.: Significance of p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:150 (July) 1941.

6. Parkinson, S. N.: Body Fluid as the Criterion for Solutions for Nasal Treatment, Ann. Otol., Rhin. & Laryng. **51**:153 (March) 1942.

therapy one should be guided by the physical structure of tissue fluid rather than by that of nasal secretion.

SUMMARY

Living tissues are perfused with body fluid, the physical properties of which are maintained at nearly constant values. Secretions and excretions on body surfaces are not body fluid but are nonliving fluids, the physical characteristics of which are uncontrolled and ever changing. Nasal mucous membrane is living tissue perfused with body fluid. Nasal secretion is nonliving fluid, the properties of which change continually as a result of many environmental factors. These changes are not to be interpreted as necessarily purposeful.

Confusion is evident in such experimen have been reported for the determinatio intranasal p_H. The cause lies chiefly in una ness that within the nasal chambers are distinct sources of hydrogen ion concentra living tissue and nonliving secretion. An i nasal electrode unavoidably contacts both sponds to both and therefore gives a comb value. Misleading, therefore, are reports situ determinations of p_H as specifically of s tion or of membrane wherein these condi have not been accounted for. By recogni of a valid premise it is hoped that experim will lead to more valid and more useful clusions.

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HYPOMETABOLISM IN THE PRACTICE OF OTOLARYNGOLOGY

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While numerous writers[1] have reported on pothyroidism in the practice of otolaryngology, vertheless, from the number of cases seen in verage otolaryngologic practice in which this dition had been diagnosed as neurasthenia, mia, hypotension, sinus disease, allergy or min deficiency I have come to believe that y otolaryngologists carry a mental picture myxedema or cretinism rather than of incipient pothyroidism. This is a mild atypical con- on that may be seen in patients of any age. It is not my purpose to discuss myxedema or tinism, for the symptoms and findings in se conditions are well known, but rather the rked variations in the clinical picture that are too frequently overlooked. In these milder es hypometabolism tends to exert a selective ion on only one—or possibly several—of the ans and systems.

Briefly, the physiology of the thyroid gland is it produces a hormone which has a definite on on the nervous system of the human being is essential to normal growth and metabolism. refore, the secretion of the thyroid gland t be maintained at a fairly constant level of duction if the human organism is to remain state of well-being.

The less obvious etiologic factors are malnutri- mental strain, worry or a sense of insecurity fe, especially as it is lived in this war period. doubt in some patients metabolism is lowered an illness which makes complete recovery ossible and more or less starts a vicious cycle. Obesity is the classic sign which usually directs ntion to hypothyroidism, but many thin per- s who never have gained weight do so when ted on a well regulated course of thyroid tment. Whereas somnolence, forgetfulness lack of concentration are signs of true myxe- a, restlessness, nervousness and insomnia are e apt to be observed with hypometabolism. dache, low blood pressure and general fatigue frequent complaints of the patients.

Blockage of the eustachian tube, with a tendency to occur and disappear in the same day, is often due to hypometabolism.[2] Inflation gives only temporary relief. Tinnitus and even degeneration of the eighth nerve may fall in this group. Many cases in which furuncles, eczema of the auditory canals or scaling and redness resist all forms of local treatment should be investigated from a metabolic standpoint.

Chronic nasal blockage, with or without a watery discharge and with or without pallor of the mucous membranes, may be secondary to hypometabolism. Postnasal drip and repeated colds in patients in whom these symptoms persist after apparently well indicated operations and in whom consideration has been given to other causes, such as allergy, diet and vitamin deficiencies, should be investigated from a metabolic angle. Often these patients have been placed in a borderline allergy group and treated with no results, or frequently they have had various cauterization procedures carried out on their turbinates.

Forman[3] pointed out that even if an allergic or a vasomotor condition is present, it is often aggravated by other factors, and hypometabolism is likely to be one of them.

Dryness of the throat, huskiness and weakness of the voice, and a sensation of a foreign body being present in the throat are frequently complained of by patients with hypometabolism. All vague pharyngeal and laryngeal complaints in the absence of pathologic change should warrant consideration of hypometabolism.

The clinical diagnosis can be substantiated by determinations of the basal metabolic rate. If on the first test the rate is within normal limits and clinically the physician is convinced of the diagnosis, several metabolic examinations should be made, as well as dextrose and galactose tolerance tests and determinations of the blood choles-

1. Bryant, B. L.: Hypothyroidism in the Practice Otolaryngology, Ann. Otol., Rhin. & Laryng. **45**: -1068 (Dec.) 1936; Thyroid Derangements: Their ct on the Ear, Nose, and Throat, J. Med. **18**:128- (May) 1937. Sprunt, T. P.: Endocrine Features nterest to the Otolaryngologist, Arch. Otolaryng. 3-77 (Jan.) 1930.

2. Drury, D. W.: Tinnitus Aurium: Its Incidence in Endocrine Disorders, J. A. M. A. **91**:1508-1511 (Nov. 17) 1928; Endocrine Hypofunction in Ear Disease, J. Laryng. & Otol. **42**:375-385 (June) 1927.

3. Forman, J.: Hypothyroidism and Vasomotor Rhinitis, Ann. Otol., Rhin. & Laryng. **43**:279-286 (March) 1934.

terol.[4] Since most metabolic tests are made on ambulatory patients, they may be counted on to record a rate at least 5 per cent higher than the rate really is.[5] A case has been reported in which the reading was a + 60. The test was repeated, with the same reading, until the patient was found to have a large perforation in the tympanum. After the auditory canal was closed with cotton, the reading was — 20.

The diagnosis of hypometabolism in a young or a mature adult does not present so difficult a problem as that of hypometabolism in a child. The differential diagnosis of cretinism is not difficult, but little is known about the milder degrees of hypothyroidism in children. Undoubtedly, many of the subnormal conditions in children are due to hypometabolism. Since normal metabolism is essential for the development of normal mentality, in those cases in which the parents seem more concerned about the child than the clinical findings warrant, the mental development, the capacity for learning, the nervousness and the irritability should be investigated. The dextrose and galactose tolerance tests, the determination of the blood cholesterol and the roentgenologic examination for delayed bony development are the best aids in diagnosing hypometabolism in children.

The contraindications to thyroid medication may be listed as follows[6]: hyperthyroidism, diabetes mellitus, serious organic heart disease, persistent tachycardia, tuberculosis and extreme degrees of malnutrition. For fear of denying to a patient who cannot improve without it the benefits of this medication, I urge that as long as there are no contraindications it be given an adequate trial even though the basal metabolic rate is within normal limits.

The treatment of patients with hypometabolism consists in the administration of any form of the prepared gland or its active principle. Various investigators have pointed out that the products of the different manufacturers vary in potency; therefore, it is well to fix on one brand and continue its use.

There are several rules for the administration of thyroid extract, but in these borderline cases it has been found most satisfactory to begin with a small amount and increase the dose gradually until the symptoms for which it is given have disappeared. During the first few weeks the patient should be seen often in order to check on possible overdosage. The amount of medication is strictly an individual problem. Throughout the literature on hypothyroidism the necessity for the patient to continue taking thyroid extract indefinitely is stressed. This is true in the cases of milder hypometabolism. After the patient is free of symptoms, the dose can be decreased gradually and the therapy finally stopped, when the metabolism has been brought to normal limits.

In reviewing our cases of the last three years I find that 138 patients had complaints or findings that suggested hypometabolism. Of the 138 patients, 25 were males and 113 were females. The average age of the males was 34 and of the females, 31 +. The chief complaint of 18 patients was headache; 26 had aural complaints; 61 had nasal complaints and 33 complained of throat symptoms. The aural complaints included vertigo as well as actual disease of the ear, and the complaints of the throat, huskiness and weakness of voice.

REPORT OF TYPICAL CASES

CASE 1.—C. F., a white woman aged 22, when first seen, Nov. 2, 1942, complained of vague pain and "tightness" over the left side of the head, not particularly localized. This sensation did not interfere with activity but was present constantly. The patient had consulted a number of physicians and was using nose drops for a "sinus condition." Gross examination of the ears, nose and the throat gave essentially negative results. Roentgen examination of the sinuses presented no pathologic change. The blood pressure was 106 systolic and 74 diastolic. The pulse rate was 78; the temperature, 98.0 F. The hemoglobin content was 80 per cent; the erythrocyte count, 5,070,000; the leukocyte count, 6,750; the Schilling count, 5/53. The Wassermann test was negative. The sedimentation rate was 0, 1 and 7. The basal metabolic rate was — 49. The patient was instructed to take small doses of thyroid extract until her symptoms were under control. She was last seen on March 4, 1943, at which time she had been entirely free of her complaint for some two weeks. At that time her blood pressure was 124 systolic and 74 diastolic; the pulse rate, 74; the temperature, 98.6 F.

CASE 2.—A. R. C., Sr., a white man aged 47, was first seen, Dec. 22, 1942, complained of impaired hearing and a stuffiness in both ears. Examination of the ears, the nose and the throat disclosed only mild hypertrophia of the nasal mucous membranes. The blood pressure was 103 systolic and 80 diastolic. The pulse rate was 66; the temperature, 97.6 F. The basal metabolic rate was — 37. The patient was given 1 grain (0.032 Gm.) of thyroid three times a day and when last seen had become completely free of symptoms after four weeks of this therapy. The blood pressure was then 118 systolic and 82 diastolic; the pulse rate, 80; the temperature, 98.0 F.

CASE 3.—L. M. W., a white man aged 45, was first seen, Feb. 29, 1944, complained of stuffiness of the nose. He stated that he had always used nose drops. He had undergone cauterization of the turbinates a number of times, with relief for from six to eight months each time. On examination, the nasal mucous

4. Rose, E.: The Diagnosis and Treatment of Thyroid Disease, M. Clin. North America **26**:1711-1737 (Nov.) 1942.

5. Kimball, O. P.: Clinical Hypothyroidism, Kentucky M. J. **31**:488-495 (Oct.) 1933.

6. Means, J. H.: Hypothyroidism: Diagnosis and Treatment, Bull. New York Acad. Med. **16**:14-19 (Jan.) 1940.

rather pale but dry and there was hyperplasia of turbinates. The blood pressure was 113 systolic and astolic; the pulse rate, 74; the temperature, 97.8 F. basal metabolic rate was —37. With thyroid py the symptoms subsided in one week. This py is still being followed, and when the patient last seen, April 21, 1944, his blood pressure was systolic and 86 diastolic. The pulse rate was 80 the temperature 98.6 F. This patient was sure that rization was his only hope for relief, and it was to persuade him to have a determination of the olic rate.

SE 4.—E. R. J., a white woman aged 34, when seen, March 5, 1943, complained of hay fever the round. She had undergone numerous allergic exations and treatments, with no results. Examinarevealed a pale, moist and swollen nasal mucosa. blood pressure was 120 systolic and 70 diastolic; the rate, 100; the temperature, 98.0 F. The basal olic rate was —24. The patient was given thyextract and in eight weeks was symptom free for irst time in three years. During the following s the treatment was discontinued, and within ty-two hours sneezing and other symptoms re-d. She was last seen in January 1944 and at that stated that she remains symptom free as long as akes 1 grain (0.06 Gm.) of thyroid every other Her basal metabolic rate at that time was —11.

SE 5.—G. C., a white man aged 32, when first March 20, 1942, complained of headache which red morning or evening and over the entire head. d persisted intermittently for ten years. With the che was associated nasal stuffiness, with mucus pharynx, and occasional acute attacks of pharyn- He had been treated for sinusitis with an allergic tion. When first seen, he had not eaten meat, bread or milk in some time. Examination of the the nose and the throat revealed a pale and boggy mucosa, with a mucoid type of discharge. His nx was injected, with granulations. Urinalysis negative results. The hemoglobin content was 80 per cent; the erythrocyte count, 5,050,000; the leukocyte count, 8,000; the Schilling test, 2/67. The Wassermann reaction was negative. The sedimentation rate was 0, 1 and 8. The basal metabolic rate was —22. Roentgen examination of the sinuses did not reveal any pathologic change. The patient was rechecked from an allergic standpoint, with essentially negative findings. The blood pressure was 112 systolic and 68 diastolic. The pulse rate was 80; the temperature, 97.6 F. A thyroid regimen was prescribed. With much effort he was persuaded that he was not allergic and that he needed to take a full diet. Within five weeks he began to show marked improvement. In September 1942 he had been symptom free for some three months; so he was told to stop taking thyroid. Immediately symptoms began to recur, which were brought under control by the resumption of thyroid therapy. The patient finally stopped taking thyroid in December 1943 and has been free of symptoms since then. The basal metabolic rate in February 1944 was —5. The patient was last seen on March 10, 1944. At that time the blood pressure was 120 systolic and 90 diastolic. The pulse rate was 88, the temperature was 98.6 F., and he was free of symptoms.

SUMMARY

Because of the negative and unimpressive character of most of the signs and symptoms of hypometabolism, the condition frequently escapes detection. Therefore, before an obscure disease is placed in the "wastebasket" of borderline allergy or neurosis, one may well investigate the metabolic status of the patient. It is amazing to observe the improvement after treatment, not only in the symptoms for which the otolaryngologist has been consulted but in the general health and well-being of the patient.

Reymond Building.

Case Reports

ADENOMA OF THE TRACHEA

JAMES A. HARRILL, M.D., WINSTON-SALEM, N. C.

Adenoma arising from the upper part of the trachea is rare. Schwartz,[1] who recorded an instance, was unable to find a report of a case of similar nature in the medical literature. My review of the available literature failed to reve similar case. It is therefore of interest to re a case of this condition in which the most pr nent clinical feature was tracheal obstructio

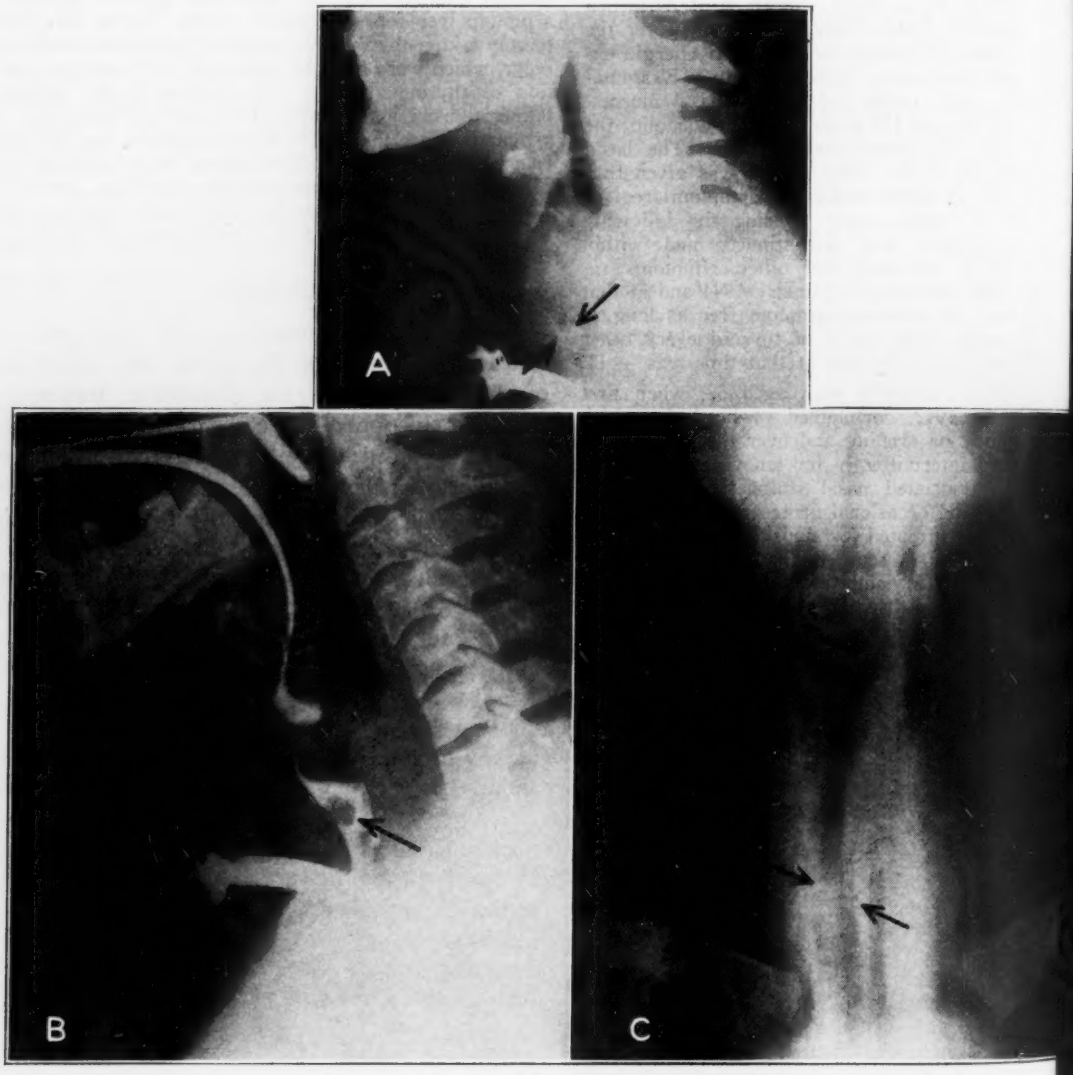


Fig. 1.—*A*, lateral roentgenogram of the soft tissues of the neck showing that the laryngeal structures normal. There is increased swelling of the retrotracheal soft tissues, with forward displacement of, and encro ment on, the posterior wall of the trachea. Within the tracheal lumen, in the subglottic region there is vi ized a small oval circumscribed opacity about 1 cm. in diameter. It appears to arise from the anterior trac wall. A tracheotomy tube is seen immediately below this tumor. *B*, lateral roentgenogram of the neck with op oil in the air passages revealing a filling defect corresponding in size and position with the mass seen i *C*, planigraphic roentgenogram demonstrating the oval circumscribed soft tissue tumor in the subglottic re

From the Department of Surgery, Division of Otolaryngology and Broncho-esophagoscopy, Bowman Gray School of Medicine of Wake Forest College.

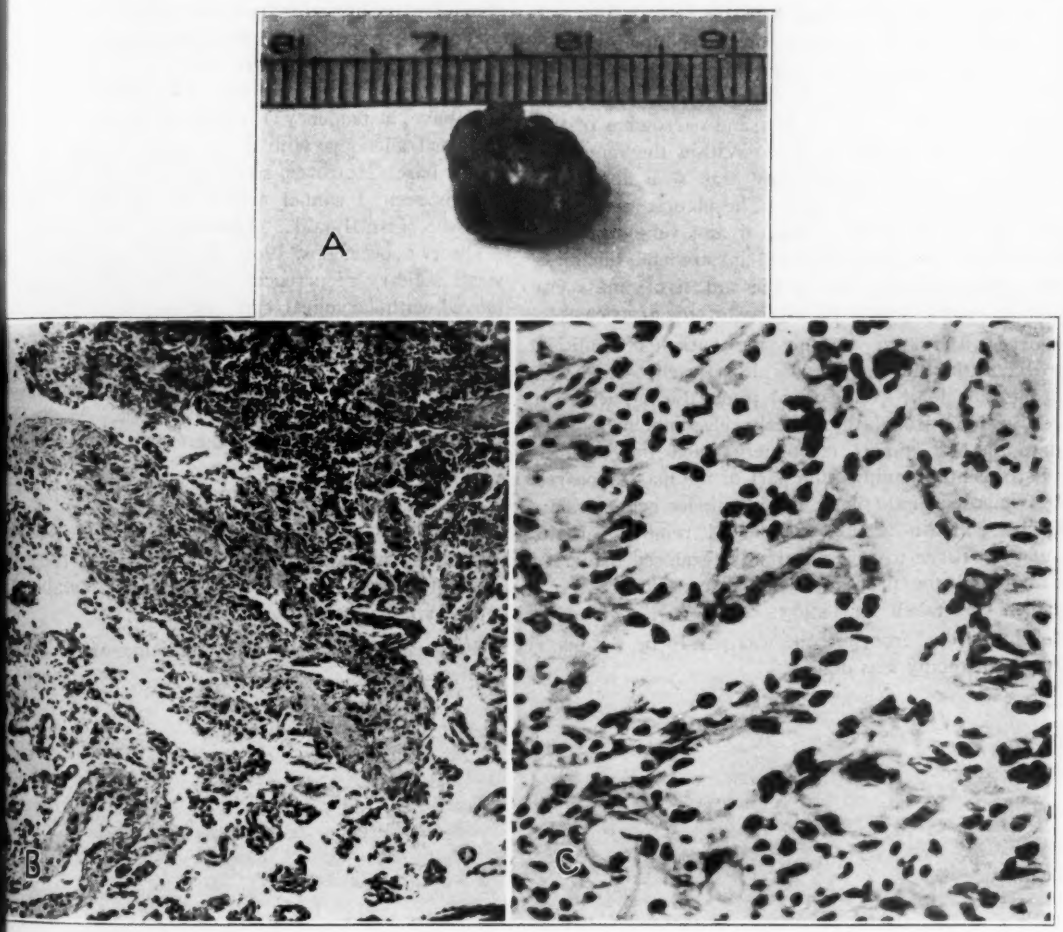
1. Schwartz, L.: Adenoma of the Trachea and the Bronchus, Arch. Otolaryng. **39**:231 (March) 1944.

REPORT OF A CASE

A 35 year old baseball umpire and former bas player was admitted to the North Carolina Baptist H pital on Oct. 16, 1942 with a chief complaint of sh ness of breath. His present illness began approxim

months previous to admission (June 1942) when as umpiring a ball game and was struck by a foul on the left side of his neck. Severe pain followed blow, and he was incapacitated for the balance of the . The skin was not broken, but moderate hemis started immediately and continued for several s. About one hour after the injury, bilateral swellof the neck was noticed, which progressed until the had become about twice the normal size. As a t of the progessive swelling, there were increasing ulty of phonation, respiratory embarrassment and ked dysphagia. He was advised by his family physito apply alternating hot and cold packs to the neck. Two months before admission he noticed some difficulty of speech and marked impairment of the singing voice.

Two weeks before admission, following an infection of the upper respiratory tract, bilateral swelling of the neck as before was noticed, which gradually increased. Respiratory embarrassment increased gradually, and the patient became progressively hoarse. Three days before being admitted to the hospital the patient collapsed while riding a bicycle. There were cyanosis, evidence of obstruction of the airway and tremendous effort in respiratory movement. On October 16, because the condition did not improve much, the patient was sent to the hospital.



ig. 2.—*A*, photograph of the tumor from the trachea. *B*, adenoma of the trachea; × 100. The tumor is comd of small mucous glands situated in a vascular connective tissue stroma. *C*, adenoma of the trachea; × 370. ular glands are seen, composed of epithelial cells which possess large hyperchromatic nuclei.

ked improvement followed their use. The hemop- lasted three to four days, and the patient was re at a baseball game several days later. Phonation dysphagia gradually improved, and the swelling ded considerably. However, the neck was never ced to its original size.

veral weeks after the accident the patient was e of the appearance and the gradual increase in oi a mass which filled the suprasternal notch. This was attached to the trachea in the midline and d rise in the neck during the act of swallowing.

A complete past medical history and a review of systems could not be obtained, owing to the patient's difficulty in answering questions. He had the usual childhood diseases. He had an attack of septic sore throat several weeks before the injury. No history of previous dysphagia or of respiratory difficulties could be obtained. Physical examination revealed (1) moderate cyanosis with respiratory distress and evident apprehension, (2) bilateral enlargement of the neck with a stony hard thyroid gland that was palpable bilaterally and in the region of the isthmus and the suprasternal

notch, (3) hoarseness and dysphagia and (4) an infection of the upper respiratory tract without evident extension to or involvement of the lungs.

Mirror laryngoscopy revealed the larynx to be moderately inflamed. The left vocal cord was in the cadaveric position. In the subglottic area about the level of the second or third tracheal ring a reddish mass protruded from the left side of the trachea and encroached considerably on its lumen. This mass was considered to be a granuloma with superimposed infection.

The temperature was 38.6 C. (101.5 F.); the pulse rate, 88; the respirations, 26; the blood pressure, 124 systolic and 64 diastolic. The results of urinalysis were negative. The hemoglobin content was 94 per cent. The red blood cell count was 5,800,000; the white cell count, 11,800, with 75 per cent polymorphonuclears.

A roentgenogram of the soft tissue of the neck revealed marked swelling of the vocal cords with obliteration of the ventricular shadow and narrowing of the subglottic region of the trachea. Within the lumen of the trachea a small oval density was seen about the level of the third tracheal ring. The planigram showed marked deformity of the laryngeal and subglottic tracheal airway due to the increase in size and thickness of the left vocal cord and to the soft tissue mass encroaching on the subglottic airway of the trachea.

Under conservative therapy the patient's condition improved little, and on October 18 a tracheoscope was passed to the level of the third tracheal ring, where a large, friable, granulating, freely bleeding mass was encountered. It almost completely filled the trachea. On instrumental manipulation part of the mass appeared to be composed of cartilage, the remainder being granulation tissue. An attempt at partial removal of the granulation tissue to give an airway produced moderate bleeding, and the procedure was discontinued after a specimen was taken for biopsy.

A 7 by 40 cm. bronchoscope was passed by the mass, and a tracheotomy was done.

The biopsy revealed that the mucosa and the ti beneath the mucosa were rather profusely infilt with chronic inflammatory cells.

Following tracheotomy, the inflammatory rea within the larynx markedly subsided, and on Octob the tracheoscope was passed down to the mass, w came away from the tracheal wall easily when gra with the forceps. The tissue removed measured 1 cm. The trachea in this area was moderately sten

The pathologic report included two opinions. Pa ogist A said: "The surface is covered by strat squamous epithelium, beneath which is a chroni inflamed area of connective tissue which is infilt with mononuclear cells. A large area of trac glandular proliferation is seen, which definitely i adenoma. At the base of the adenomatous area the vary in size and shape, are hyperchromatic and pr an occasional mitotic figure. The glands are irre and have a tendency toward infiltration. Diagn Tracheal adenoma with a low grade malignant ch in the base. However, since only a small portion o base is seen, I cannot arrive at a definite conclus

Pathologist B said: "Adenoma of the trachea."

On two occasions, Nov. 24, 1942 and Dec. 2, small solitary recurrences were observed and were moved with laryngeal cup forceps.

In view of the questionable malignant change at base of the original tumor and the presence of the change in the two recurrences, 1,680 roentgens were given to the anterior part of the neck. At the time writing, six months later, there has been no evid of recurrence.

SUMMARY

In the case of tracheal adenoma present dyspnea, wheezing and stridor were the pro nent features. The tumor was probably tr matic in origin.

Bowman Gray School of Medicine of Wake For College.

OCCULT THROMBOSIS OF SIGMOID AND LATERAL SINUSES

An Unusual Case

MAJOR A. HARRY NEFFSON, MEDICAL CORPS, ARMY OF THE UNITED STATES

The case to be presented is instructive from standpoint of diagnosis, operative findings therapy.

REPORT OF CASE

April 27, 1943, while swimming, the patient, a ur old white soldier, was kicked in the right ear. night pain in the ear and some loss of hearing oped. The past history was entirely noncontrib-; he had never had trouble with his ears before. patient was in excellent health. The general al examination gave entirely normal results. ly a purulent discharge from the right ear and rhinitis were noted.

was given ten 1 Gm. tablets of sulfadiazine and o take four tablets a day, spacing them at four als, until they were gone.

next day there was less discharge, and two days the ear was dry. During the next ten days he seen in the outpatient clinic every other day be- of mild nasopharyngitis. During this time the as dry and the soldier was on full duty.

May 17 (twenty days after the onset of the s) he returned to the clinic; at this time there a scant discharge from the ear, with edema, red- and tenderness of the external canal, and some uricular edema but no tenderness over the mas- rocess. A diagnosis of external otitis and recur- otitis media was made, a wet gauze pack was ed into the canal, a roentgen examination of the id processes was made and he was admitted to ospital. The next day the roentgen report stated both mastoid processes were sclerotic, with no nce of mastoiditis. A wet dressing was applied e ear. During the next three days there was al improvement in the external otitis, but there still a scant discharge coming from the right e ear. A day later (the twenty-fifth day follow- the onset of the aural discharge), his temperature to 103 F., and he complained of severe right-sided ache. Examination showed a slight discharge in ear, no tenderness over the mastoid process but tenderness over the region of the emissary vein. e was no involvement of the facial nerve and no gmus. There was a slight amount of nuchal ity. A spinal puncture was done, and clear fluid, under pressure, was obtained. This showed 91 lymphocytes per cubic millimeter; a smear dis- d no bacteria. The administration of sulfadiazine started; the dosage was 1 Gm. every four hours. next morning there was a slight chill and the erature was 104 F.; a blood culture was taken. patient stated that he felt better and had less ache. There was no increase in rigidity of the There was a slight discharge from the ear. chest was normal both on auscultation and on gen examination. The eyegrounds were normal. e were no petechiae. A diagnosis of thrombosis e sigmoid sinus was made, but because his tem- ture remained high (around 105 F.) all day, ation was deferred till the next morning, when it was down to 100 F. The sulfadiazine was increased to 2 Gm. every four hours.

Operation and Findings: The usual incision over the mastoid process was made, the skin and the periosteum were retracted, and the cortex was elevated with mallet and gouge. A few small cells filled with mucoid material were found in the initial groove. The rest of the mastoid process was completely sclerotic. The antrum was opened widely. The plate overlying the sigmoid sinus was normal looking and extremely hard; it was elevated by means of mallet and gouge. The sinus was found to be discolored and thickened. The sigmoid sinus was then exposed from the region of the bulb up to the "knee," without finding normal-appearing sinus. The lateral sinus was then exposed as far back as ½ inch (about 1 cm.) from the torcular Herophili. The sinus appeared to be somewhat less discolored at this point, but it did not look normal. Iodoform gauze plugs were inserted at both ends near the bulb and the torcular. The sinus was then incised and found to be thickened and full of thrombus. With a grooved director and scissors the entire length of the exposed sinus was opened and a thrombus about 4 inches (10 cm.) long was removed. Following this, there was rather brisk bleeding, apparently from the region of the petrosal sinuses. This was controlled with iodoform gauze packing. Sulfanilamide powder was placed in the wound, more iodoform gauze was inserted, and the wound was closed with several loose sutures.

The patient's condition was such as to make it unwise to perform ligation of the jugular vein at this time. He had been under anesthesia induced with sodium pentothal for a period of over two and one-half hours. It was decided to wait and see whether a chill developed and then to ligate the jugular vein. He was given a transfusion of 500 cc. of citrated blood and 250 cc. of plasma during the operation. Later he was given 5 Gm. of sodium sulfadiazine and 1,000 cc. of 10 per cent dextrose in isotonic solution of sodium chloride intravenously.

The next day his temperature was still spiking, and toward evening he had a chill. He was again given an additional 5 Gm. of sodium sulfadiazine intravenously. The blood culture was reported as showing Streptococcus haemolyticus beta; the culture of the spinal fluid showed no growth. On the following day he looked weaker; his heart frequently dropped beats, but the sounds were of good quality. His temperature was still spiking, but he had no chills. His blood count, which four days previously had been 8,000 white cells, 80 per cent of which were polymorphonuclear, now dropped to 4,000 white cells with 58 per cent polymorphonuclear leukocytes. In the fear that this might be due to a toxic reaction from the chemotherapy an attempt was made to obtain penicillin, but this was unsuccessful because at that time it was available for use only at certain general military hospitals. He was in no condition to be transported several hundred miles, and therefore the treatment with sulfadiazine was continued in the hope that the leukopenia was caused by the infection rather than by the drug.

On the third postoperative day his condition was still precarious; his temperature was 103.6 F., and he complained of a headache again, but there had been no chills. On June 1, the seventh day after operation, he complained of pain, and there was tenderness on the right side of the neck. He was taken to the operating room, and the iodoform plug was removed from the bulbar end; there was no bleeding. A rubber catheter was inserted into the bulbar end of the sinus, and it was irrigated with warm isotonic solution of sodium chloride. The return was clear. Pressure on the jugular vein produced no response. On removing the iodoform packing at the upper end, there was brisk bleeding, probably from the superior petrosal sinus. The plug near the torcular was not removed at this time because of this bleeding. The wound was repacked and dressed. The sulfadiazine was increased to 3 Gm. every four hours for the next twenty-four hours, and then it was given at the rate of 2 Gm. four times a day. During this time the blood count rose to 19,900 white cells, with 76 per cent polymorphonuclear leukocytes. Sulfadiazine was continued at a dosage of 1 Gm. every four hours. Vitamins A, B complex and D were also given in suitable doses. On June 5 both the packing and the torcular plug were removed; no bleeding followed. Sulfanilamide powder was placed in the wound, an iodoform gauze drain was inserted, and the wound was closed.

From this time on his condition progressively improved. As has been noted by others, the growth of granulations here too was definitely depressed because of the use of sulfanilamide powder in the wound. The sulfadiazine was continued in progressively decreasing doses. Altogether he received 265 Gm. of sulfadiazine by mouth and intravenously over a period of six weeks, with no ill effects. The blood level during this time averaged 9 to 10 mg. per hundred cubic centimeters; the low level was 4.0 mg. and the high 14.4 mg. per hundred cubic centimeters.

He was discharged to full duty after a ten weeks' stay in the hospital and a furlough of one month for convalescence. When last seen—one year after his illness—he was in excellent health, the wound was closed and looked good, and his hearing was normal.

COMMENT

This case is reported because of the diagnostic problem which it presented, the unusual findings during operation, the prolonged anesthesia induced with sodium pentothal, the large amount of sulfadiazine used, the question of early discontinuance of this therapy, and uneventful recovery even though a normal was not encountered at the torcular end the jugular vein was not ligated.

The diagnosis of thrombosis of the sig sinus was made on the basis of the aural i tion, the tenderness over the region of the sary vein, the chill and the dural irritation denced by the presence of an increased nu of lymphocytes in the spinal fluid. The that the soldier had been ambulatory and there was little infection of the ear, no cli or roentgen evidence of mastoiditis and petechial eruption was disturbing, but not suading. Although phlebitis of the sig sinus was expected, the extent of the inv ment in a patient who had been ambulatory fever free until two days previously was astonishing. This necessitated the use of so pentothal for a period of nearly three h much longer than had been contemplated. increase of the operative time was due to hardness and thickness of the skull and lack of suitable instruments and trained a tants. The ominous drop in the white b cells and the polymorphonuclear leukocyte the second day after operation led to an att to obtain penicillin. Since this was not avail I decided to continue the treatment with s diazine, hoping that the depression of the le cytes was due to the severity of the infe rather than to the toxicity of the drug. F nately for the patient, this turned out to b case, since he was able to tolerate 265 Gm. of s diazine over a period of six weeks, never manifesting any toxic reaction. The jug vein was not ligated because further manip tion during the operation had been deemed wise and later he had had only one chill, w occurred on the first day after operation.

I feel that the patient's life was saved by use of sulfadiazine. Early diagnosis and op tion, no doubt, helped.

THROMBOSIS OF THE CAVERNOUS SINUSES

MAJOR WILLIAM C. WOLFE
MEDICAL CORPS, ARMY OF THE UNITED STATES
AND
CAPTAIN JOHN F. GAIN
SANITARY CORPS, ARMY OF THE UNITED STATES

Prior to the advent of chemotherapy, recovs in cases of thrombosis of a cavernous sinus e extremely rare. In a recent article MacNeal [1] marized reports of 58 cases in which there recovery, covering a period of many years edical literature. His article places particular hasisis on the staphylococcus as the causative nism in this condition and includes an excel- review of the anatomic and the pathologic ervations and the intracranial pathways and ins, as well as the case reports. He also dis- es the various forms of treatment that have advocated, including surgical operation, in- ion of serum, and the use of bacteriophages sulfonamide compounds. Since the case we reporting falls into one of the groups he has ussed, no further résumé of these aspects will given.

REPORT OF A CASE

white man aged 49 was admitted to the Army and y General Hospital, Hot Springs, Ark., on Oct. 31, . He was acutely ill, unconscious and irrational. first history was obtained from his wife. Five days to his admission a small furuncle developed on ip of his nose; in two days it became large, swollen painful. His local physician prescribed hot packs sulfathiazole, 60 grains (3.88 Gm.) per day. There no attempt at incision or manipulation. On this men his illness rapidly progressed. Twenty-four s before admission he had a severe chill and became tional. His past history was noncontributory.

ysical Examination on Admission.—The patient well nourished and hydrated but appeared acutely his temperature was 104 F.; his blood pressure 130 systolic and 80 diastolic. There was a fairly demarcated red inflamed area of the skin over eyelids and the regions of the cheeks and forehead nd the eyes. There were marked chemosis, prop- and fixation bilaterally but more marked on the The motion of the left eye was limited to about legrees; that of the right eye was somewhat less ed. The right fundus showed definite venous en- gement, and the left could not be seen because of the e induration which prevented opening of the lids. e were no other findings of note on initial exam- on.

oentgenologic and Laboratory Examinations.—Roent- studies of the skull and sinuses on November 9 November 11 showed nothing of significance. The n test was negative. The blood culture yielded a growth of Staphylococcus aureus haemolyticus. On ssion, a blood count showed 4,800,000 red cells per c millimeter, with 16 Gm. of hemoglobin per hundred cubic centimeters, and 28,000 white cells, with 90 per cent polymorphonuclears. The bleeding time and the clotting time were normal, and the prothrombin level was 54 per cent of normal. The urine showed albumin (4 plus) and an occasional pus cell. For ease of correlation, further laboratory data and the schedule of penicillin therapy are summarized in table 1. As an aid in determining the proper therapy, the effects of penicillin and of sulfadiazine on the growth of this organism in vitro were studied. These studies are described in the section of the protocol devoted to bacteriologic observations.

Progress.—Intramuscular injections of penicillin were started, 2 cc. (10,000 units) being given every hour for four hours and thereafter 2 cc. every three hours. The blood prothrombin level on admission was 54 per cent of normal, probably on the basis of impaired function of the liver, as will later be discussed; neither heparin nor dicoumarin (3,3'-methylene bis [4-hydroxycoumarin]) was used. The possibility that these drugs might be used to inhibit further thrombosis was constantly considered, but the prothrombin level remained low. Twenty-four hours after admission the temperature had fallen to between normal and 101 F.; the patient was restless but rational. There was marked rigidity of the neck, and a spinal tap revealed very cloudy fluid. Penicillin was given intrathecally at the rate of 20,000 units a day for four days. The patient appeared distinctly jaundiced, and there was marked distention of the urinary bladder. A retention catheter was installed. Throughout the first few days of the patient's illness he had periods in which he was irrational but he was always sufficiently cooperative to take fluids well by mouth. A high intake and output could be maintained. On the third day after admission, because of a drop in the red blood cell count, a transfusion of 500 cc. of citrated blood was given. By the fifth hospital day there was definite clinical improvement. The spinal fluid had cleared from 1,135 cells to 35 cells; culture of the blood gave a negative result on this day for the first time and at no time gave a positive result during the remainder of the patient's period of illness. There was recession of the local findings about the eyes, with motion returning in the right. The nonprotein nitrogen of the blood and the icteric index remained high, the nonprotein nitrogen level reached its highest, 99 mg. per hundred cubic centimeters, on the twelfth hospital day, while the icteric index declined steadily after admission, reaching approximately normal on the twenty-sixth hospital day.

On the sixth day of hospitalization the dosage of penicillin was dropped to 10,000 units every four hours, and this was continued through the ninth hospital day. Through this period clinical improvement continued, particularly as to jaundice and toxemia. The temperature became normal, and the patient was completely rational. On the tenth day, with a slight elevation in temperature, the patient appeared slightly less well oriented and mentally alert than on the previous day. There were no definite neurologic signs, but the clinical picture at this point was considered suggestive of a

1. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: ombophlebitis of the Cavernous Sinus, Arch. Ophth. 231-257 (Feb.) 1943.

localizing abscess of the brain or an expanding extradural abscess. Roentgenograms of the skull made to exclude osteomyelitis showed none. The patient continued semistuporous for three days, during which time the temperature was elevated only slightly. The dosage of penicillin was reduced to 10,000 units every six hours. Neurologic consultation revealed no findings other than moderate rigidity of the neck. At this time the neurologic consultant stated: "The picture is considered to be undoubtedly one of marked impairment of brain function, but it cannot be determined whether this is on the basis of infection and toxicity or an actual destructive lesion of the brain. Since the former could account for the entire clinical picture there is no positive evidence at this time for any local lesion in the brain."

On the fourteenth day of hospitalization a definite rash appeared over the neck, thorax and abdomen, apparently of a drug type, and the administration of penicillin was discontinued.

For the next twenty-three days there was steady improvement. The nonprotein nitrogen declined from 99 mg. on November 12 to 33 mg. per hundred cubic centimeters on November 26. The icteric index fell from 67 on November 2 to 9.2 on November 26. The urine cleared, and the specific gravity remained within normal limits. There was clinical improvement commensurate with the laboratory picture. The patient was rational, well oriented and cooperative; on a soft diet, he ate well and maintained excellent nutrition. During this period the only finding was persistent papilledema of about 3 diopters and fairly marked venous engorgement of both fundi. The chemosis and proptosis subsided and motion of both eyes returned. Vision on gross testing was approximately normal bilaterally. The patient was allowed out of bed for short periods each day.

TABLE 1.—*Results of Laboratory Studies*

	Blood						Blood Chemistry							
Date	Red Blood Cells, Millions per Cu. Mm.	Hemoglobin, Gm. per 100 Cc.	White Blood Cells, Thousands per Cu. Mm.	Bleeding Time	Clotting Time	Prothrombin Level, per Cent of Normal	Nonprotein Nitrogen, Mg. per 100 Cc.	Urea Nitrogen, Mg. per 100 Cc.	Creatinine, Mg. per 100 Cc.	Total Protein, Gm. per 100 Cc.	Icteric Index	Sugar, Mg. per 100 Cc.	Carbon Dioxide, Volumes per Cent	Chlorides, Mg. per 100 Cc.
Oct. 31	4.8	16	28	40″	4′	..	..	..	....	....	..	....	..	
Nov. 1	....	....	....	...	...	54	..	..	....	....	..	...	..	
2	4.12	12.5	29	...	...	58	55	..	2.4	....	67	125	..	
3	3.66	12	29.7	...	...	..	70	..	2.2	....	61	138	..	
4	5.16	14.5	32.5	...	...	..	69	..	2.15	....	58	...	61	
5	3.98	12	22	...	...	..	66	..	1.8	....	53	...	..	
6	4.05	12.5	24.9	...	...	..	66	..	1.8	....	28	...	..	
7	4.2	13.0	30.4	...	...	..	76	..	1.9	....	21	...	..	
8	4.62	13.5	28.9	...	...	73	83	..	1.9	....	22	...	..	
9	4.3	13	27.9	...	...	86	83	..	1.85	....	19	...	..	
10	4.61	13.5	33	...	...	..	93	..	1.95	....	23	...	..	
11	4.7	14	35	1′15″	2′30″	..	83	..	....	6.8	25	...	..	
12	4.6	13.5	35	...	...	..	99	57	....	....	22	...	..	
13	4.31	13.5	24	...	...	..	83	54	1.8	....	25	...	..	
14	....	....	....	...	...	..	77	..	....	....	21	...	..	
15	4.5	13.5	15.3	...	...	..	67	..	1.5	....	18	...	..	
16	....	....	....	...	...	..	..	..	....	....	..	...	..	
17	....	....	....	...	...	..	55	..	....	....	14	...	..	
18	....	....	....	...	...	..	..	..	....	....	..	...	..	
19	....	....	....	...	...	..	58	..	....	....	13	...	..	
20	....	....	....	...	...	..	..	..	....	....	..	...	..	
21	....	....	....	...	...	..	..	..	....	....	..	...	..	
22	....	....	....	...	...	..	..	..	....	....	..	...	..	
23	4.6	13.5	8.5	...	...	..	44	..	....	....	13	...	..	
24	....	....	....	...	...	..	..	..	....	....	..	...	..	
25	....	....	....	...	...	..	..	..	....	....	..	...	..	
26	4.0	12.5	8.8	...	...	..	33	..	....	....	9.2	...	..	
27	....	....	....	...	...	..	..	..	....	....	..	...	..	
28	....	....	....	...	...	..	..	..	....	....	..	...	..	
29	....	....	....	...	...	..	..	..	....	....	..	...	..	
30	....	....	....	...	...	..	..	..	....	....	..	...	..	
Dec. 1	....	....	....	...	...	..	..	..	....	....	..	...	..	
2	....	....	....	...	...	..	28	..	....	....	8.1	...	..	
3	....	....	....	...	...	..	..	..	....	....	..	...	..	
4	....	....	....	...	...	..	..	..	....	....	..	...	..	
5	....	....	....	...	...	..	..	..	....	....	..	...	..	
13	3.98	11	21.5	...	...	..	21	..	....	....	7.8	...	..	
14	....	....	....	...	...	..	..	..	....	....	..	...	..	
15	3.8	12	18	...	...	..	..	..	....	....	..	...	..	
16	....	....	....	...	...	..	..	..	....	....	..	...	..	
17	3.94	12	29	...	...	..	..	..	....	....	..	...	..	
18	4.34	....	20	...	...	..	..	..	....	....	..	...	..	
19	....	....	....	...	...	..	..	..	....	....	..	...	..	
20	....	....	....	...	...	..	..	..	....	....	..	...	..	
22	....	....	....	...	...	..	..	..	....	....	..	...	..	
23	....	....	....	...	...	..	..	..	....	....	..	...	..	

On December 5, the thirty-fifth day of hospitalization, there was a slight elevation of temperature, and the patient complained of a frontal headache. This continued for the next few days, with the temperature reaching 101 F. daily. During this time the patient became apathetic and lethargic. He responded to commands, but sluggishly. There were no neurologic findings until December 12, when a slight facial weakness on the left referable to the central nervous system was noted, together with a questionable weakness of the left arm and the left leg.

On December 13 definite localizing signs were present. There was marked weakness of the lower part of

the arm and the leg, with hyperactive reflexes, on left side, as well as a sustained left ankle clonus. ectile vomiting appeared for the first time. The eye showed 2 diopters of papilledema and mild us engorgement; the left showed 3 diopters of lledema and marked venous engorgement, plus a e-shaped hemorrhage temporally.

n December 14 there was complete left-sided spastic iplegia. There were pain and tenderness over the temporal bone. Roentgenograms showed a definite of a well calcified pineal body to the left of the ine. There was no change in the pulse or in the

maintained with procaine hydrochloride. A transfusion of 500 cc. of blood was given on the operating table. The immediate postoperative condition was fairly good. The deep stupor, present before operation, persisted. There were several frank shaking chills during the day, and the rectal temperature averaged about 103 F. There was marked nuchal rigidity. Penicillin was given again, 10,000 units every three hours, and its administration was continued on this schedule during the remainder of the patient's life in the hospital.

The postoperative course was gradually downhill. The stupor persisted, the patient being in coma from

the Schedule of Penicillin Therapy

	Urine				Spinal Fluid					Penicillin Therapy			Fluid Balance	
lood ulture	Specific Gravity	Albumin	Pus Cells per High Power Field	Phenolsulfonphthalein, per Cent Excreted	Cell Count	Polymorphonuclears, per Cent	Smear	Culture	Sugar, Mg. per 100 Cc.	Intramuscular, Units × 10^3	Intraspinal, Units × 10^3	Transfusions, Cc. Citrated Whole Blood	Intake, Cc.	Output, Cc.
....	1.017	4+	Occ.	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	90	..	...	3,600	700
Pos.	.....	3+	0	..	1,366	76	White blood cells	Neg.	45	80	20	...	2,000	.....
Pos.	1.013	3+	Occ.	..	1,135	73	White blood cells	Neg.	..	80	20	500	3,600	3,200
Pos.	1.014	2+	0	..	35	4	.....	Neg.	..	80	20	...	4,000	2,900
Neg.	1.012	1+	6-7	..	155	98	Red blood cells	.....	..	80	20	...	4,900	3,500
....	1.010	0	Many	..	.....	..	.....	.....	..	60	..	...	4,600	3,100
....	1.007	1+	5-7	..	.....	..	.....	.....	..	60	..	...	4,500	3,000
....	.....	...	....	..	.....	..	.....	.....	..	60	..	...	4,500	3,400
Neg.	1.013	0	0	..	42	92	Neg.	Neg.	..	60	..	...	3,600	2,400
....	1.013	Trace	4-8	..	.....	..	.....	.....	..	40	..	...	4,500	4,000
....	1.012	Trace	0	..	.....	..	.....	.....	..	40	..	...	4,400	2,500
....	1.011	Trace	4-8	..	9	8	Neg.	Neg.	..	40	..	...	6,000	3,700
Neg.	.....	...	....	50	.....	..	.....	.....	..	55	..	...	4,900	3,100
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	5,300	2,000
Neg.	.....	...	....	..	.....	..	.....	.....	..	..	..	...	3,900	2,000
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	3,700	2,100
Neg.	1.008	Trace	Many	..	.....	..	.....	.....	..	..	..	...	3,700	2,100
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	4,700	3,000
Neg.	.....	...	....	..	.....	..	.....	.....	..	..	..	...	4,000	2,700
Neg.	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	1.004	0	Many	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	1.003	1+	Many	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	Trace	1-3	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	0	Many	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	1.005	0	0	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	1.004	0	4-8	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	..	..	...	.....	.....
....	1.014	1+	0	..	.....	..	.....	.....	..	..	..	...	.....	.....
Neg.	1.014	1+	0	..	.....	..	.....	.....	..	80	..	...	.....	.....
....	1.018	1+	1-2	..	0	..	Neg.	.....	..	80	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	80	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	80	..	...	.....	.....
....	1.017	0	0	..	.....	..	.....	.....	..	80	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	80	..	...	.....	.....
Neg.	.....	...	....	..	.....	..	.....	.....	..	80	..	...	.....	.....
....	.....	...	....	..	.....	..	.....	.....	..	80	..	...	.....	.....

d pressure. The patient rapidly became more orous. The neurosurgical consultant made a diagis of abscess of the right frontal lobe of the brain. t operation, on December 15, a large abscess was d, deep in the right frontal lobe. The brain was soft and necrotic, and there was apparently little psulation. A large amount of pus and necrotic n tissue was evacuated. Direct smear and culture his revealed Staph. aureus of a hemolytic type. soft rubber catheter surrounded by gauze drains ed in penicillin was inserted into the abscess cavity anchored to the edges of the scalp wound. The sthesia for the entire operation was induced and

the day prior to operation until his death on the ninth postoperative day. The spinal fluid pressure varied from 350 to 420 mm. of water, and daily spinal taps were required to relieve it. The fluid was grossly clear, and on laboratory examination there were no significant findings. Nutrition and hydration were maintained by a nasal feeding tube. Repeated blood cultures were sterile. A heavy purulent exudate continued to discharge from the wound. The patient died on December 24 at 6:40 p. m.

Autopsy.—Only the pertinent observations will be described here. The left lung was boggy. Slight crepitance was demonstrable anteriorly. The bronchi con-

tained much yellow mucopurulent fluid. Their mucosal surfaces appeared light grayish red and edematous. The cut surface of the lung presented a striking appearance. The upper lobe appeared bright grayish red, speckled with numerous opaque grayish green spots 2 to 4 mm. apart. These spots were rather uniformly spaced. In the posterior portion of the lobe were several cavities, 1 to 2 cm. in diameter. These contained thin greenish gray fluid. Their walls were thin, showing little evidence of fibrous tissue. The lower lobe contained practically no air. Numerous opaque grayish green spots resembling those in the upper lobe were scattered throughout the lower lobe. Air could be demonstrated in the alveoli only in the extreme anterior portion. The appearance of the cut surface of the lower lobe was essentially quite similar to that of the upper lobe except that the entire surface appeared tinted with the green material forming the prominent opaque spots. Several cavities were found in the posterior portion of the lower lobe. These were similar to those in the upper lobe except that the exudate they contained was somewhat thicker and darker in color.

The right lung was also very boggy and there was slight crepitance anteriorly. The bronchi contained tenacious mucopurulent material which in places coated the grayish red, edematous bronchial mucosa. The pleural surface was dull bluish gray, tinted with green posteriorly. On the inferior surface several cavities, averaging 1 cm. across, opened onto the pleural surface. These were surrounded by grayish brown fibrinous strands. They contained thin greenish gray exudate and resembled the cavities in the left lung. Much turbid grayish red fluid could be expressed from the cut surface. Posteriorly, several cavities were present in the upper lobe.

The heart and all abdominal viscera were essentially normal. There was moderate generalized arteriosclerosis.

The brain weighed 1,360 Gm. The operative incision opened into a large cavity lined with soft grayish red tissue. The brain was adherent to the dura over the superior and anterior portions of the right frontal lobe. When the dura was separated from the brain in this region, a large quantity of thick pea-green exudate was noted along the courses of the superficial cerebral vessels on each side of a large subdural abscess. On the right side the dura was also adherent over the superior and anterior portion of the parietal lobe. Beneath the dura in this location was a similar collection of pea-green exudate which was in communication with the anterior portion of the abscess. On removal of the dura and the exudate, irregular depressions of the cerebral cortex were observed. These formed the inferior walls of the abscess and were covered with adherent grayish white fibrinopurulent exudate. Anteriorly, these depressions involved most of the superior frontal gyrus and the posterior portion of the middle frontal gyrus. A narrow depressed area in the extreme posterior portion of the superior frontal gyrus communicated with the posterior portion of the depressed abscess. This involved the superior portions of the anterior and posterior central gyri. The depression of the cerebral cortex was at most 3 cm. in depth.

The falx cerebri was displaced to the left. The operative incision entered the cerebral cortex slightly below the lowest point of the large anterior portion of the subdural abscess on the right side; thus the incision did not enter the subdural abscess. The opening of the incision in the cortex was located in the superior part of the posterior end of the inferior frontal gyrus on the right side. The incision communicated with a l irregular cavity lined with reddish gray necrotic tissue. On the right side, in the superior frontal g at the level of the genu of the corpus callosum abscessed cavity 2 to 3 cm. in diameter was This abscess was filled with thick pea-green exu resembling that of the subdural abscess. There however, no visible fibrous or fibrinous wall surro ing this abscess, which consisted simply of a colle of exudate in the brain tissue. On the right si similar abscess was noted somewhat farther poster being at the level of the truncus of the corpus callo This abscess was also thin-walled and filled with pea-green exudate. Laterally this abscess was ab but not quite in communication with the large c draining through the operative incision. The ventr were of normal size and contained clear fluid. Nu ous thin coronal sections were made, and the inte markings were noted to be everywhere normal. left and right cavernous sinuses were removed. T appeared similar. Their lumens could not be dem strated grossly, so that their locations were ma only by a small amount of brown material in thin la of fibrous tissue surrounding the internal ca arteries. The posterior intercavernous sinus appe as a firm cord about 2 mm. in maximum diameter necting the two cavernous sinuses. The anterior i cavernous sinus was very small. The superior sag sinus and the great cerebral vein were both patent.

Microscopic Observations.—The lungs showed typical features of multiple pulmonary abscesses. other viscera were essentially normal. Sections thro the right and left cavernous sinuses appeared essenti similar. The lumen was completely obliterated by l connective tissue. Through this ran large numbers dilated thin-walled capillaries. The aggregate cr sectional area of all these approximated only a frac of the size of the original lumen of the sinus. Thro the loose connective tissue were scattered large numb of lymphocytes and macrophages filled with gol brown pigment. Here and there were small quanti of bright pink-staining persistent fragments of fi Many of these had rounded outlines and were obvio undergoing dissolution. Polymorphonuclears were frequent. Numerous sections of the brain and m showed the changes typical of pachymeningitis, me gitis, extradural and cerebral abscesses. There w marked necrosis and congestion throughout.

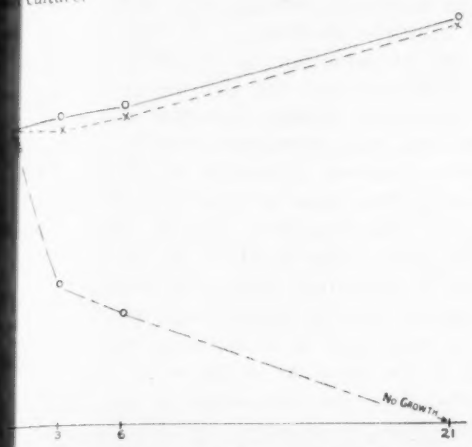
Bacteriologic Study.—On the isolation in pure c ture of Staph. aureus of the hemolytic type from original blood culture in vitro, studies were made the bacteriostatic effect of sodium sulfadiazine and p cillin on the micro-organism. The protocol of t studies follows:

1. An aqueous solution of penicillin containing 5 Oxford units per cubic centimeter was diluted with p broth so that 0.5 cc. of solution contained 0.06 un the drug. With this dilution being used as the hig concentration, serial dilutions in plain broth were m in a series of eight tubes.

2. Aqueous solutions of sodium sulfadiazine w prepared and diluted so that 0.5 cc. contained sod sulfadiazine in the concentration of (1) 25 mg. hundred cubic centimeters and (2) 5 mg. per hun cubic centimeters.

3. For purposes of control, a tube containing 0.5 of plain broth was included in the test.

Each of the aforementioned tubes was inoculated 0.5 cc. of a (1 : 5,000,000) dilution of an eighteen second broth subculture of the original positive culture.



cteriostatic effect of penicillin and sodium sulfadiain vitro. The graphs represent curves of bacterial s on cultures as follows: a plain broth culture as ol (——); a broth culture containing sodium sulfae in the concentration of 25 mg. per hundred cubic meters (– – – –); a broth culture containing peni- in the concentration of 0.06 unit per 0.5 cubic cenr (— – —).

After three, six and twenty-one hours' incubation C., 0.1 cc. was removed from each of the various and diluted with plain broth. Poured plates for ts of bacterial colonies were made in duplicate from al of these dilutions.

e accompanying figure graphically shows the re- of the experiment. For convenience, only the ts for the tube containing that amount of penicillin showed complete bacteriostatic action (0.06 unit ubic centimeter) and those for the tube containing igher concentration of sodium sulfadiazine were d.

though bacterial counts were not made, a tube conng sodium sulfathiazole in the concentration of 25 er hundred cubic centimeters, which was tested in ilar manner, showed no inhibition of growth of est organism.

om an in vitro standpoint it is evident that neither e sulfonamide compounds would have been effective mbating the septicemia.

autopsy the purulent material in the brain yielded re culture of Staph. aureus of the hemolytic type, the lung was found to contain a streptococcus of emolytic type.

experiment similar to the former but with higher ntrations of a freshly prepared solution of peni- was set up. The results are summarized in table 2.

COMMENT

The case of thrombosis of the cavernous sinuses reported presents several unusual features. It is of particular interest from a bacteriologic standpoint. Pure cultures of a hemolytic strain of Staph. aureus were recovered from the primary lesion of the skin and from the blood. This organism was proved in vitro to be resistant to sulfonamide compounds but very responsive to penicillin. This corroborates the work of Crowe and his co-workers.[2] Under large doses of penicillin the septicemia was controlled and the patient showed marked clinical improvement, with resolution of the classic features of thrombosis of the cavernous sinus. Despite the sterilization of the blood stream, however, this same organism invaded the brain and the dura extensively, with formation of multiple abscesses. Concurrently, under intensive penicillin therapy, there was extensive terminal pneumonia with multiple abscesses of the lungs, the causative organism here being a hemolytic streptococcus found in pure culture at autopsy. Thus, the penicillin which was proved effective clinically and in the laboratory against a hemolytic strain of Staph. aureus was ineffective against this particular hemolytic strain of Streptococcus. It also had no effect on organisms within an abscess cavity, as is true of all chemotherapeutic agents.

TABLE 2.—*Bacteriostasis Exerted on the Isolated Organisms by Penicillin in Vitro*

of penicillin per cubic centimeter of culture	2	1	0.5	0.25	0.125	0.0025	0.03	0.015	0.0075
nce of bacterial growth in:									
eart's blood (staphylococcus)*.....	0	0	0	0	0	0	Trace	+	+
rain (staphylococcus)*	0	0	0	Trace	+	+	+	+	+
ung (hemolytic streptococcus)*.....	0	Trace	+	+	+	+	+	+	+

A plus sign means that the culture was turbid. "Trace" indicates that there was no turbidity but that growth was ed on subculture. A zero means that there was no growth on subculture.

Sufficient equipment was not available for studies of the concentrations of penicillin in the blood stream during therapy. If the work of Rammelkamp and Keefer[3] on the absorption and excretion of penicillin may be used as a guide, it is most probable that the dosage used would produce concentrations in the blood serum sufficient to counteract the original septicemia. However, it is apparent from the increased amount of penicillin necessary to inhibit the growth of the staphylococcus in the brain culture that the organism was developing some resistance to the action of the drug. The increased resistance in vivo is comparable to that

2. Crowe, S. J.; Fisher, A. M., and Ward, A. T., Jr.: Penicillin and Tyrothricin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **52**:541-561 (Sept.) 1943.

3. Rammelkamp, C. H., and Keefer, C. S.: The Absorption, Excretion and Distribution of Penicillin, J. Clin. Investigation **22**:425-437 (May) 1943.

noted in vitro.[4] Because of the larger amount of penicillin necessary to produce bacteriostasis in the brain culture, it is possible that, had the patient lived longer, he would have again had septicemia.

The comparatively large amount of penicillin necessary to inhibit the growth of the hemolytic streptococcus isolated from the lungs at autopsy is strong evidence that the patient might have died from his streptococcic pneumonia even had there been no other pathologic process if treated only with penicillin, since it is highly improbable that a blood serum level of 0.5 unit per cubic centimeter was ever obtained. It is also of interest that no apparent bacteriostasis was exerted on this organism by either sulfadiazine or sulfathiazole when these drugs were tested in concentrations of 25 mg. per hundred cubic centimeters.

Despite the large quantity and the prolonged use of penicillin, the only complication encountered was a rash, not severe, which cleared promptly when the administration of the drug with discontinued. Lyons [5] stated that urticaria was present in 5.7 per cent of his cases and that it is not regarded as important. The intrathecal use of penicillin caused no symptoms or reactions.

Probably the most interesting feature of this case from a bacteriologic standpoint is the dramatic proof in vitro of the ineffectiveness of the sulfonamide compounds in combating this hemolytic strain of Staph. aureus. This observation was corroborated clinically by the fact that the patient had been treated with sulfathiazole for five days prior to his admission to the hospital, with no effect.

The very high values for nonprotein nitrogen and the icteric index can be explained only by the severe toxemia incident to the sepsis, with marked suppression of hepatic and renal function. At autopsy there were no morphologic changes in these viscera.

On the basis of the alterations observe autopsy and the clinical course, we believ is safe to say that this patient had thromb of the cavernous sinuses with meningitic pye and multiple abscesses. All this was cured the use of penicillin, as proved by the reces of symptoms and the definite recanalization the cavernous sinuses observed at autopsy. hold that the subsequent death of the pat does not detract from the effectiveness of pe cillin in this type of infection but rather e phasizes that this drug, like all others, has m limitations. Above all, it is not a specific must be used only when the indication is c firmed by careful bacteriologic study.

In retrospect it is apparent that this pat was in the invasive stage of an abscess of frontal lobe on admission to the hospital, within five days after the development of initial lesion, a furuncle on the tip of the n This stage was evidenced by a chill, toxe stupor and a high spinal fluid cell count. the time it could not be determined that th symptoms were not due to the thrombosis of cavernous sinuses and the bacteremia. Whe earlier use of penicillin would have influen this rapid spread and invasion of the infec organism is problematic.

SUMMARY

A patient with thrombosis of the cavern sinuses was treated with penicillin. On the ba of the clinical and laboratory studies it see probable that penicillin is the only chemothe peutic agent available that would have be effective against the causative organism, hemolytic strain of Staph. aureus. Autopsy su stantiated the clinical diagnosis and the reco of the clinical course.

Major Fritz Cramer, Kennedy General Hospital, P formed the surgical operation in this case; Lieute Colonel Irving S. Wright and Major Alfred Pinker of the Medical Service, Army and Navy General H pital, supervised the administration of penicillin. Ma Joseph E. Flynn and Lieutenant Frank Mann perfor the autopsy.

4. Schmidt, L. H., and Sesler, C. L.: Development of Resistance to Penicillin by Pneumococci, Proc. Soc. Exper. Biol. & Med. **52**:355-357 (April) 1943.

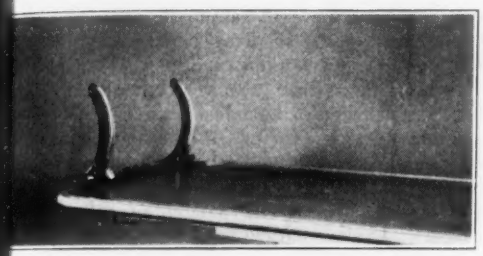
5. Lyons, C.: Penicillin Therapy of Surgical Infections in the U. S. Army, J. A. M. A. **123**:1007-1018 (Dec. 18) 1943.

Clinical Notes

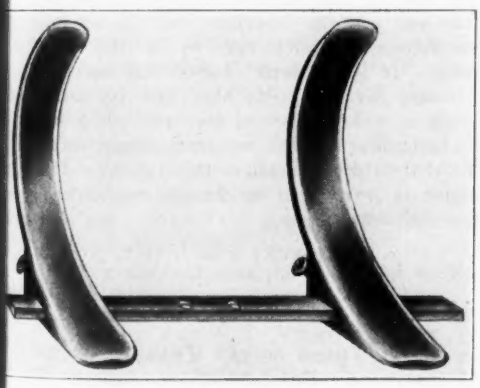
TWO AIDS TO BRONCHOSCOPIC EXAMINATION

HOWARD E. CRAWFORD, M.D., HILO, TERRITORY OF HAWAII

e devices to be described were conceived by Miss
. Middleton, director of nurses at Puumaile Hos-
and have been found of such value in carrying out
choscopic examinations that their use should be
rted.



g. 1.—Device for holding the patient's shoulders
and in place while the head is elevated for
choscopy.



g. 2.—Diagram showing details of the set-up in
re 1.

he usual bronchoscopic team calls for a second
stant to hold the shoulders of the patient so that the
d may be elevated without the shoulders being raised.
only does this require more help but this assistant
difficulty in preventing the shoulders from sliding
to one side or the other. To obviate this and the need of a second assistant the following adaptation was made.

A metal bar of proper width and thickness to fit the slots in the shoulder braces commonly used on surgery tables is bolted to the end of the table which we use for bronchoscopy. The convex surface of the braces is covered with sponge rubber. They are then applied to the metal bar in the reverse position (figs. 1 and 2). The patient is moved to the head of the table; he places his arms around the braces and is slid downward until the braces are firmly engaged in the axillas. The braces can be adjusted to any desired width but should be tight against the thorax. While we use a hydraulic spinal

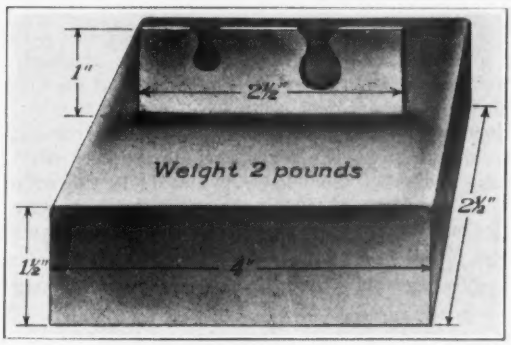


Fig. 3.—Diagram showing the structure of a device for holding the suction tube and the light cord in place on the instrument table.

stretcher for bronchoscopy, the appliance can be fitted to other tables as well.

The second device is used to hold the suction tube and the light cord in place on the instrument table. It consists of a block of wood to which is attached an upright piece of metal with slots in it of proper size to hold the tube and the cord. These slip in and out easily. The wood is completely covered with solder to hold the metal strip in place and to give weight up to 2 pounds (about 1 kg.), which is sufficient to prevent the block being moved by the drag of the tube (fig. 3).

Abstracts from Current Literature

Ear

PNEUMATIZATION OF THE ADULT TEMPORAL BONE, THE MASTOID PORTION: AN ANATOMIC AND CLINICAL STUDY. J. D. SINGLETON, Laryngoscope **54**:324 (July) 1944.

Intimate knowledge of the various cell groups of the temporal bone aids in enabling the surgeon to make a clean and thorough exenteration of the cells in the mastoid process.

The anatomy and the relations of the mastoid process are discussed. The reader is reminded that the mastoid portion of the temporal bone is made up of three parts: the petrosa, the squama and the annulus.

Pneumatization may be confined to the mastoid antrum or it may extend into the squama. The petrosa may be entirely pneumatized. Both the occipital bone adjacent to the petrosa and the petrous tip may be invaded by cell groups.

The author divides the cells into those of the squama and those of the petrosa, and divides these groups into various subgroups of cells, which he describes in detail. It is noted that a septum is frequently found between the squama and the petrosa. Septums may also separate many of the various subgroups of cells from one another.

The signs and symptoms of mastoiditis differ according to which cell groups are particularly involved. A correlation between the groups involved and the clinical pictures is presented.

Roentgenographic examination is of great importance not only for diagnosis but for locating the various cell groups and other landmarks invaluable to the surgeon. The actual surgical approach and procedure may be varied to advantage in relation to the presence and the extent of the various cell groups.

HITSCHLER, Philadelphia.

TRAUMATIC DEAFNESS: PROBLEMS OF PREVENTION. H. MARSHALL TAYLOR, Laryngoscope **54**:362 (July) 1944.

Taylor points out that the eighth nerve is the most easily injured of any of the nerves of the special senses and has poor powers of recuperation. The human ear has not adapted itself biologically to the intense noises that occur in some phases of present day life. The war has greatly increased the number of ears exposed to high intensities of sound.

Noise causes degeneration of certain of the hair cells of the organ of Corti. The degeneration does not remain stationary but spreads later to other hair cells.

Cotton plugs have been and are being used to prevent acoustic trauma. These plugs, which reduce the noise by but a few decibels, are almost worthless. Plugs or defenders made of rubber or plastic are much more effective. The author hopes that the latter will come into general use in the military services.

HITSCHLER, Philadelphia.

CONTRIBUTIONS TO FUNCTIONAL PATHOLOGY OF THE EAR: I. ON SOME ASPECTS OF TREATMENT IN PROGRESSIVE CONGENITAL DEAFNESS. F. KOBRAK, J. Laryng. & Otol. **58**:439 (Nov.) 1943.

Kobrak suggests that the activity of the intratympanic musculature is significantly changed in congenital deafness and that this change is due to a spastic con of the muscles secondary to primary pathologic ch in the cochlear nerve. In this series of observa treatment was directed toward altering the abn condition of the cochlear nerve and also that o motor branches of the fifth and eighth nerves supp the intratympanic muscles and the proprioceptive n of the muscles.

A report of 31 cases of otosclerosis and coc deafness is presented, in which the author did systematically use strychnine and arsenic, as he feels secondary degeneration of the nerve occurs after administration of these drugs is stopped, giving p hearing than before treatment. It is reported calcium and phosphorus are beneficial when used gether. In this series, vitamin D was also given times with calcium. Vitamin B was used in cases because of the reported beneficial effect in sp conditions in other regions.

Kobrak believes that it is important to eliminate substances which are harmless in a normal perso dangerous to the hearing of the patient with congenital deafness. Among these substances tobacco and the salicylates, a common offender b acetylsalicylic acid. The danger of intense noi the ear of the patient with otosclerosis is emphasized.

He admits that it is not possible to alter the pathologic changes in congenital deafness. The pati condition may be improved, however, by fenestratin labyrinth, thus making a new pathway for sound w or by altering two variable factors, the pressure i middle ear and the labyrinth and the activities o intratympanic muscles and so of the damping paratus. It is the latter factor that concerns hi the main, for he holds that the tympanic mu operate at a key position for controlling normal pathologic hearing and to some degree encourage medical treatment of congenital deafness. The gre promise is offered in combating the early stage congenital deafness.

LEJEUNE AND STEELE, New Orlea

Pharynx

THE MIXED TUMOR OF THE TONSIL AND THE V PALATINUM. ERNESTO RAFFO, Rev. de cir., **1**:101, 1943.

Raffo reports a case in which a man 42 years o complained of a sensation of fulness in his throat had no difficulty in swallowing either solid or l food and was free of pain or discomfort. Examin of his throat revealed a large mass in the left ton fossa. This was removed in toto, with the region u local anesthesia, and there were no untoward a effects of the operation and no recurrences. The hi logic examination proved the mass to be a mixed tu

PERSKY, Philadelph

LYMPHOSARCOMA OF THE TONSIL. BENJAMIN KA Rev. de otorhinolaring. **3**:222 (March) 1944.

Kaplan presents 3 cases of lymphosarcoma o tonsil. Radiation therapy was employed with va results. One patient recovered fully. One died.

has extensive metastasis and is in a critical con-
n.

ncer of the tonsil usually occurs between the ages
0 and 60. It grows insidiously, often without pain
with but slight discomfort. It metastasizes to the
ical lymph glands fairly early. As a rule, it is
ly radiosensitive and in consequence responds rapidly
radiation. The prognosis is favorable if the lesion
eated early, especially when there is no metastasis.

PERSKY, Philadelphia.

Larynx

CE AND BREATHING DISABILITIES FOLLOWING THYROID SURGERY. CHARLES GORDON HEYD, New York State J. Med. **44**:1905 (Sept. 1) 1944.

r. Heyd, who is professor of surgery at the New
k Postgraduate Medical School and Hospital, read
paper before the Southern Surgical Association at
Orleans in December 1943. He lists the vocal
culties after operation on the thyroid gland as
ws: (1) spasmodic cough without expectoration,
retention of mucus and inability to clear the throat,
pain over the molar teeth and in the ear, (4)
heal aspiration ("swallowing the wrong way"), (5)
fatigue of a normal voice, (6) change in pitch of
e, (7) continual hoarseness, (8) inspirational stridor,
stridor and "crooning" when sleeping, (10) whisper
e only, (11) dyspnea on exertion, (12) dyspnea on
slightest exertion and (13) dyspnea when at rest.
anatomy, especially the innervation of the larynx,
eviewed. According to the extent and the site of
y, one may have partial or complete paralysis of
cord or complete abductor paralysis of both cords
called separator paralysis) with crowing and dif-
t inspiration, which may call for tracheotomy.
d says that all of these untoward symptoms lessen
time, usually within three months. Dr. Charles
Fiertz has successfully treated some 30 patients by
dy cathodal galvanization over the nerve and
rrupted stimulation over the laryngeal muscles.
object is to get the vocal cords to move. This
require from six to eight weeks. A case of bi-
ral abductor paralysis of seven years' duration is
rted. The patient had had no, or insufficient, treat-
t. Galvanization removed the vocal and breathing
bility after thirty-seven treatments.

VOORHEES, New York.

Nose

US DISEASES AND NATURAL DEFENCES. J. G. WHITAKER, J. Laryng. & Otol. **58**:397 (Oct.) 1943.

ecause of the ability of certain substances, such as
ssium iodide and pure broth, to induce an increased
x of leukocytes when in contact with the peritoneal
ty, Whitaker feels that they would be of value in
ting for chronic sinusitis since phagocytosis is the
dition sine qua non of natural defense. He has
d that the flow of phagocytes, especially the
rophages, in the sinuses is even more rapid than
in the peritoneum after instillation of a solution of
ssium iodide. It is felt that stimulation of phago-
sis is necessary only in chronic sinusitis, since in
e sinusitis the presence of purulent nasal secretions
ts to the occurrence of phagocytosis. Chronicity is
ght to be caused by a disturbance in capillary
meability which tends to prevent the passage of
ocytes, seemingly a more important factor than poor drainage. The capillary wall must have only a functional change since it allows excitation of phagocytosis; if irreversible lesions are present, surgical intervention is advisable.

The method of treatment is outlined. First the sinus ostiums are shrunk by instillation of a solution of equal parts of 3 per cent ephedrine and 3 per cent cocaine; then the potassium iodide in 8 to 10 per cent solution is caused to enter the sinuses by the displacement method of Proetz. An application of Brüning's light bath is then made to the head for five to seven minutes. It is thought that this helps to stimulate phagocytosis. The treatment outlined is performed every other day, and after an average of eight treatments, it is stated, improvement approaching cure is procured. If after the third day of treatment, poor progress is being made, a solution of histamine phosphate is instilled by the Proetz technic prior to the instillation of the solution of potassium iodide, so that the potent capillary dilative effect of this substance can be brought to bear. Suction is advantageous for dilating capillaries as well as for mechanically removing pus.

As a study preliminary to the method of treatment described, 151 cases examined at autopsy are reported, in 86, or 56.9 per cent, of which sinus disease was shown. The ethmoidal sinuses were diseased in 63 cases, the maxillary sinuses in 58 and the frontal sinuses in 29. The diseases with which sinusitis was found to be most frequently associated were cardiovascular disease, tuberculosis and pneumonia. The prevalence of sinusitis was greater in persons between the ages of 20 and 40 years.

LEJEUNE and STEELE, New Orleans.

PLASTIC SURGERY OF THE NOSE. ALBERTO FLORES, Rev. de cir., Lima **1**:77, 1943.

Flores discusses the present role of plastic surgery, and in emphasizing the importance of this specialty, stresses the fact that it truly belongs to the rhinologist rather than to the general surgeon. He describes the normal anatomy of the nose with some of the common deviations. His surgical procedure is presented in an outline which, while superficial, is still sufficient to convey the surgical principles usually employed by the rhinoplastic surgeon. His technic is essentially that of Joseph. He presents a series of before and after photographs which illustrate his excellent results. In conclusion Flores stresses the fact that a good rhinoplastic surgeon must possess a certain amount of artistic temperament to obtain the better result.

PERSKY, Philadelphia.

Miscellaneous

HIGH FREQUENCY DEAFNESS AND DISCRIMINATION OF "HIGH FREQUENCY" CONSONANTS. ROBERT N. PLUMMER, J. Speech Disorders **8**:373 (Dec.) 1943.

A test of aural discrimination involving fifteen so-called high frequency consonants was administered to 399 college students. From this group 52 persons were selected to represent a scale of distribution from poorest to best. Nine of the 52 students were found to have loss of hearing for high frequencies. Three had general loss of hearing. The 9 persons with loss of hearing for high frequencies had no appreciable difficulty in discriminating between the high frequency consonants; the 3 with general loss had such difficulty. Discrimination of the sounds of specific consonants did not appear to

be dependent on sensitivity to specific frequencies in the higher ranges.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

SUBDURAL EMPYEMA. CHARLES S. KUBIK and RAYMOND D. ADAMS, Brain **66**:18 (March) 1943.

"Subdural empyema" denotes a collection of pus in the subdural space. Kubik and Adams report 14 cases. In 12 cases the condition followed infection of the paranasal sinuses, in 1 case it was secondary to infection of the ear, and in 1 case it was metastatic from a chronic suppurative lesion in the lung. In 12 cases the outcome was fatal, and in 2 there was recovery; in both the latter the cerebral abscess resulted from infection of the sinuses. Thirteen of the 14 patients were males, and 8 of the 14 patients were between the ages of 12 and 30 years, the ages of the others varying up to 55 years. Subdural empyema usually follows an acute episode of a chronic infection, though in 1 of the reported cases it was apparently secondary to primary acute sinusitis. Chronic frontal sinusitis with frontal osteomyelitis and epidural infection is particularly favorable for its development.

The onset is marked by local pain which is referred to the region of the affected ear or sinus, followed by generalized severe persistent headache with nausea and vomiting. This is accompanied by a pronounced rise in temperature. The increased temperature is associated with increased intracranial pressure and rigidity of the neck. Sooner or later the patient becomes confused or somnolent, the condition progressing rapidly to stupor or coma. At about the same time focal neurologic signs begin to appear, the most important being jacksonian seizures (seen in 50 per cent of cases), hemiparesis, aphasia and paralysis of contralateral conjugate deviation of the eyes. In the fatal cases death occurred two to five days after the appearance of focal signs. Roentgenograms usually give evidence of sinusitis and, in some instances, of frontal osteomyelitis. The cerebrospinal fluid is under moderately increased pressure, and there is pleocytosis of a moderate degree, with predominance of polymorphonuclear cells. Bacteria were never demonstrated in smears or by culture.

With the onset of focal signs, sinus thrombosis and cerebral abscess must be differentiated. In the former condition there is seldom a stiff neck, and the signs are bilateral. Cerebral abscess has a milder, more insidious onset, and the course is usually more protracted. The temperature is seldom high, and the cell count of the spinal fluid is normal or only slightly increased; lymphocytes predominate. In 4 of the 12 cases of subdural empyema complicating sinusitis the subdural abscess occurred by direct extension through the dura following osteomyelitis of the frontal bone; in 2 cases it followed necrosis or perforation of the bony wall of the frontal sinus or of the ethmoid cells and dura, and in 1 case it followed an operative fracture. In the remaining cases it followed thrombophlebitis of a venous sinus. The subdural exudate usually covered a la part of one cerebral hemisphere. The exudate thickest over the lower lateral portion of the fro lobe, extending to the orbital and medial surfaces, the temporal lobe and as far back as the parietal l It was never walled off. A subarachnoid exudate limited to the area covered by the subdural collec There was rarely generalized leptomeningitis.

Microscopic studies revealed thrombosis or thron phlebitis of the adjacent subarachnoid veins and se ischemic necrosis of the subjacent cortex.

Treatment consists of surgical drainage as soo the diagnosis is made. Drainage is best accompli through an enlarged burr hole in the lateral fro region, since this is the usual site of the gre accumulation of exudate. This approach provide attack through a clean field and results in no har exploration gives negative results. The dura ov subdural abscess is white and easily recognized. use of sulfonamide compounds is recommended.

SHENKIN, Philadelphia.
[ARCH. NEUROL. & PSYCHIA

ON THE SO-CALLED "LARYNGEAL EPILEPSY." C. W. WHITTY, Brain **66**:43 (March) 1943.

Whitty reports 4 cases of laryngeal epilepsy, a c dition occurring in middle-aged men of plethoric t in association with laryngitis or bronchitis. A pre personal or family history of epilepsy is uncom The presenting symptom is an attack of dizziness loss of consciousness, preceded by a tickling sens in the larynx and cough, which may be slight or se Epileptiform movements may occur during the at but a more constant feature is slight mental confu following the attack. In about one fourth of the c apparent recovery or remission follows local treatm of the upper respiratory tract.

The author found that forced expiration with a cl glottis failed to produce asystole or convulsions in of his cases. That cerebral venous congestion anoxemia due to cough were etiologic factors was garded as unlikely, since venous constriction failed produce an attack in any case in which forced coug produced an aura. Furthermore, it was found tha a case in which venous obstruction produced an aura attacks were independent of coughing. The author c cludes, therefore, that in the majority of cases the c dition is primarily neurogenic and represents a f of genuine epilepsy.

The neurogenic factor which appeared to be at in the cases reported was true reflex laryngeal epile epilepsy with a laryngeal aura or venous conge caused by violent coughing in a case in which cerebral cortex was predisposed to epilepsy, whe by vascular degeneration or by other factors.

SHENKIN, Philadelphia.
[ARCH. NEUROL. & PSYCHIA

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

Sixty-Sixth Annual Meeting, New York, June 7 to 8, 1944

CHARLES J. IMPERATORI, M.D., *President*

ARTHUR W. PROETZ, M.D., *Abstracter*

Morning Session

Wednesday, June 7, 1944

sidential Address. By DR. CHARLES J. IMPERATORI, New York.

cocele in Frontal and Ethmoidal Sinuses: Simplified Surgical Treatment. Presented by DR. H. M. GOODYEAR, Cincinnati.

ucocele of the paranasal sinuses most frequently inves the ethmoid cells and the frontal sinuses. Since early symptoms are frequently double vision and lling of the inner canthus of the eye, the patient visit the ophthalmologist first and in many cases rtunately is operated on by an external approach ugh the orbit. Dr. Goodyear held that this approach rong and emphasized the approach through the nose, ch is made by a comparatively simple operation. mucocele is lined with good epithelium, and its ents are usually sterile. By opening a way through nose the lower surface of the mucocele is removed, the duct leading into the frontal sinus, which is d with epithelium, is not disturbed.

r. Goodyear showed four slides illustrating typical s.

DISCUSSION

r. Francis L. Weille, of Boston, asked if the speaker had any experience with multilocular mucoceles.

r. Ralph Fenton, of Portland, Ore., questioned the ticability of this procedure in the case of a large cele extending over the roof of the orbit and often pying a large part of the frontal sinus. He said in such a case it would be better to use the exal approach.

r. George B. Wood, of Philadelphia, asked whether the etiologic standpoint one should consider a cele as a cystic dilatation of some mucous gland s the result of occlusion of the duct, for it is impor- to know whether the mucocele might be present out completely filling the sinus.

r. Harris B. Mosher, of Marblehead, Mass., agreed Dr. Fenton on the advisability of opening the al sinus when it is invaded by a mucocele. He agreed with the essayist that nature's epithelization he cavity is far superior to any lining which the eon could produce. Dr. Mosher pointed out the eme size these growths may attain, citing 2 recent s in which exophthalmos and displacement of the were marked and in which he felt that the frontal s was to a large degree involved.

r. Goodyear, in closing, remarked that he had never a multilocular mucocele, although he assumed that a condition might occur. As a rule, the intranasal approach does not permit a full view of the entire cystic cavity.

If the external approach cannot be used, he said, it is not wise to remove the lining which Nature has provided, and he would therefore be opposed to dissecting out the sac.

Commenting on Dr. Wood's question, Dr. Goodyear expressed the belief that the mucocele has its origin in a superior or an anterior ethmoid cell in which the mucosa undergoes changes whereby the glands expand and by pressure extend into adjacent cells and the frontal sinus.

Despite Dr. Fenton's and Dr. Mosher's comment, the author felt that if the mucocele is adequately opened intranasally and the passage kept patent, one frequently will find that the external deformity will quickly disappear. At any rate, he has been able to reduce it by simple pressure on the thin distended orbital plate.

The Local Use of Sulfadiazine, Radon, Tyrothricin and Penicillin in Otolaryngologic Practice. Presented for DR. S. J. CROWE, Baltimore, by DR. DONALD F. PROCTOR, Baltimore.

The cardinal principles of successful chemotherapy are: All infecting organisms, both aerobes and anaerobes, must be identified; the bacteria must be sensitive to the drug or the combination of drugs used, and, finally, the drug must come into direct contact with bacteria in the tissues in an adequate concentration and over a sufficient period to allow complete bactericidal or bacteriostatic effect. In acute spreading infections, sulfonamide compounds given by mouth or penicillin injected intravenously or intramuscularly are usually most satisfactory, but in treating chronic infections of mucous membrane or bone much depends on the accessibility of the infecting bacteria to the action of the drug. For example, Dr. Crowe has often seen pneumococcic meningitis secondary to chronic infection of the ear or the frontal sinus temporarily improve following the administration of sulfonamide compounds or the intravenous, intramuscular or intrathecal injection of penicillin, only to recur again and again until the primary focus was sought out and drained surgically.

The antibacterial substance penicillin is produced by only a few of the many hundreds of strains of the mold Penicillium notatum. Fleming announced the discovery of penicillin in 1929, but more than a decade elapsed before its chemotherapeutic possibilities were fully realized. Since 1940 many reports on methods of preparation, purification and clinical application of penicillin have been published in England and in this country, but with few exceptions these studies have been directed toward the use of penicillin in the treatment of pneumonia, meningitis, septicemia and other systemic infections. It is not yet possible to make penicillin synthetically, and the available supply of the sodium or calcium salt of penicillin for intramuscular or intravenous injection is still limited, but crude or unrefined penicillin can be made in any bacteriologic laboratory and is often effective for local application.

Otolaryngologists have an exceptional opportunity to develop the local use of antibiotic substances in the treatment of infection, since most of the organisms in

infections of the ear and the upper air passage are gram positive and sensitive to the action of one or more of these drugs.

Acute infections that are not walled off by the natural defenses of the body and those extending to the meninges or the blood stream must be treated by saturating the entire body with antibacterial drugs, but the local use of these substances to aid in the cure of an infection of a sinus, an ear or the pharynx has been ignored with the exception of powdered sulfonamide compounds, which are usually applied at the conclusion of an operation, with no knowledge of the identity of the infecting bacteria or of their sensitivity or resistance to these drugs.

The text in this paper is limited to the following topics: 1. The local use of a solution of sulfadiazine to inhibit the growth of pyogenic organisms in the nose, the nasopharynx and the pharynx and thus prevent many of the infections of ears and sinuses complicating the common cold. 2. The local use of tyrothricin or of crude penicillin in the treatment of acute and chronic infections of the ears and upper air passages. If the conditions of successful chemotherapy mentioned are fulfilled, it is possible to cure an acute infection of a maxillary sinus with one irrigation, or to sterilize a wound so completely that following an extensive surgical operation for acute osteomyelitis of the frontal sinus and skull the incision heals by primary intention with no redness, swelling, suppuration or other evidence that the wound has become infected. 3. The local treatment of hyperplastic lymphoid tissue in the nasopharynx for the prevention or the cure of an impairment of hearing secondary to partial or complete obstruction of the eustachian tubes, for the prevention of recurring colds with aural or sinal complications and for alleviating asthmatic bronchitis in some patients, especially children.

DISCUSSION

Dr. Thomas E. Carmody, of Denver, asked what amount of radium was used, since only the duration of the treatment was mentioned. He questioned whether cessation of symptoms after one irrigation could properly be called a cure, since he had seen the same result in years past regardless of what irrigating agent was used.

Dr. Howard Ballenger, of Winnetka, Ill., reported good results from the use of powdered sulfathiazole in acute nasopharyngitis. The powder has the advantage of remaining in contact with the tissues for a longer period than the solution and therefore need not be applied so often.

Dr. John McLaurin, of Dallas, Texas, stated that he had used radium and radon in small capsules threaded back through the end of a nasal cannula, but doubted whether this procedure destroys all of the lymphoid tissue. Careful examination had convinced him that islands of this tissue persist even after irradiation and continue to act as foci of infection. To destroy these completely might at the same time cause damage to normal tissue, which has a definite value in the resistance against future colds.

To a question of Dr. Harold I. Lillie, of Rochester, Minn., whether controls had been used against the author's series, Dr. Proctor replied that there had been controls.

Dr. DeForest C. Jarvis, of Barre, Vt., described his experience with apple penicillin in mother of vinegar, which can be taken by mouth, applied locally or given by vaporization. He has seen striking results from its use in infections in live stock. He also has found it effective in furuncles of the auditory canal, eczema, fever (by inhalation), streptococcic sore throat, acute rhinitis. He recalled that the Duke of Wellington had given vinegar to his soldiers as a source of vitamin and that long before that, the Roman legions used vinegar to prevent fevers and putrefaction.

Dr. Bernard J. McMahon, of St. Louis, inquired how long the treatment is continued and whether, following the use of penicillin, there is any tendency to recurrence of these infections of the upper respiratory tract.

Dr. Proctor, in closing, described a new type of radium applicator now being used under Dr. Crowe's supervision, which is made of monel metal, is smaller in diameter than the original model and is capable of holding 50 mg. of the salt. The dosage of one treatment is 5.5 milligram hours, i. e., 50 mg. for six to one-half minutes on each side, with 1 mm. of metal filtration, which filters out the beta rays. The treatments are given at intervals of four to six weeks over a long period. The average number of treatments given the ordinary child at the Johns Hopkins Hospital has been 3.4 per child, although in 1 case a large amount of lymphoid tissue was still present after nine treatments. Dr. Proctor stated that in most cases the lymphoid tissue can be eliminated and claimed to have demonstrated this fact in hundreds of cases. However, if there is an infection in the tonsils or the sinuses radium therapy will be ineffective unless the focus is removed.

Although they have used sulfonamide compounds in powder form a great deal, they believe they get better results from the sulfadiazine solution, which penetrates the mucosa, whereas the powder does not. As for the single irrigation curing acute sinusitis, the essayist stated that in these cases the patient is followed closely and the sinuses irrigated again to make sure. After radical surgical procedures on the frontal or the maxillary sinuses, irrigations are continued for a week or ten days and then at longer intervals until the solution persistently returns clear.

Replying to a question of Dr. Burt Shurly, of Detroit, Dr. Proctor stated that tyrothricin was used in a 1:1,000 alcoholic solution, 1 cc. of which was diluted with 100 cc. of sterile distilled water. At operation, however, penicillin is used with equal parts of water.

Traumatic Deformities of the Nasal Septum. Presented by DR. SAMUEL SALINGER, Chicago.

Trauma of the nose in children should receive careful attention; dislocated septal cartilage should be replaced, hematoma evacuated and spreading of the nasal bones prevented by proper application of an external molded splint.

The primary care of the injured septum should include hemostasis, replacement of fragments, careful smoothing out of torn mucosa and proper packing. These must be coordinated with the treatment of the other concomitant injuries.

Deformities resulting from inadequate primary care must be corrected with due regard for the necessity of preserving such cartilage as is necessary to the support and the contour of the nasal bridge and tip.

Slides were shown illustrating typical cases, details of technic in certain types of deformities and methods of splinting.

DISCUSSION

Dr. Thomas E. Carmody asked whether in using the Metzenbaum technic the essayist ever found it necessary

move some cartilage in order to get it into the
e of the vomer.

Salinger, in closing, stated that he appreciated
act that the procedures depicted were subject to
fications at the hands of various surgeons to meet
ic indications.

ere are times when it is necessary to remove a part
e obstructing cartilage, but the important objective
e preservation of sufficient cartilage in the lower
ted portion to sustain the tip of the nose.

Afternoon Session

Wednesday, June 7, 1944

hing Otolaryngology in Wartime. By CAPTAIN
. P. SCHENCK, M.C., U.S.N.R. (Read by title.)

dergraduate instruction has been maintained at a
level in spite of increased classes, reduction of the
year to a three year course and release of many
ers to the armed forces. Clinical faculties in the
alties are largely volunteer, the younger teachers
in military service and the ranks of the older
ers being depleted by death and infirmity at a
increased by the burden of extended teaching
ules and mounting private practice. Deterioration
rsonal instruction has thus been inevitable. Eco-
c factors have decreased the teaching material
erly available to outpatient departments. Training
e specialties during internship and residency has
red because of less time for contacts with patients,
tion of medical and nursing staffs and lack of
rtunity to develop sound professional habits. Or-
ed graduate training is almost nonexistent. There
present provision for the training of a continuous
of teaching personnel. In each crop of medical
nts the several hundred who would normally con-
their preparation for careers in medical teaching
research are lost. Those engaged in medical
gogy must form positive plans for the postwar
d in order to minimize a serious loss to medical
ation.

sinusitis: A Résumé. Presented by LIEUTENANT
OLONEL P. A. CAMPBELL, Medical Corps, Army of
e United States.

rial operations at high altitudes and the use of
de chambers in the oxygen indoctrination program
led to an increase in the incidence and the im-
nce of aerosinusitis. Aerosinusitis results from a
ure differential between the inside and the outside
sinus following a change in the environmental
metric pressure, most often in descent. Of the
us complications of aerosinusitis, the mucosal hema-
is probably of most importance. Histologically,
nsists of an accumulation of blood and fibrin be-
n the loose tissues of the periosteum and those of
mucosa. It may be demonstrated roentgeno-
hically. After a period of a few weeks to a few
ths the hematoma in most cases resolves. Recovery
aerosinusitis usually follows equalization of pres-
between the inside of the sinus and the external
onment, regardless of the method by which equali-
n is accomplished.

DISCUSSION

r. Ralph Fenton expressed appreciation of Colonel
pbell's research in aviation medicine and the benefit
will ultimately accrue to civilian medicine as it has
far in the service. He has noted the discomfort complained of by civilians who are forced to travel by plane at considerable heights in crossing mountain ranges and the danger of complications, or at least of exacerbation, among those suffering from sinusitis. Dr. Fenton inquired of Colonel Campbell whether the use of oxygen is of any particular value in obviating disagreeable results of altitude flying.

Dr. Robert F. Ridpath, of Philadelphia, asked whether the essayist had noted any increase in the subjective symptoms during ascent or descent, particularly as to headache, pain and vertigo.

Dr. Thomas C. Galloway, of Evanston, Ill., mentioned the work done at Northwestern University by Professor Ivy and Dr. Greenwood, with regard to the therapeutic effect of negative pressure in patients with acute disease of the respiratory tract and sinusitis (Butler, D. B.; Greenwood, G. J., and Ivy, A. C.: Reduced Atmospheric Pressure as a Form of Treatment for Paranasal Sinusitis, ARCH. OTOLARYNG. **40**:266-274 [Oct] 1944). Gratifying results are reported to have been obtained in more than 50 per cent of the cases. These results would seem to be at variance with the effects observed by Colonel Campbell, although Dr. Galloway admitted that in the experiments at Northwestern University the "descent" in the pressure chamber was accomplished at a rate much lower than that in the instances cited by the Colonel.

He also said he would like to know why there seems to be a difference between the Army and the Navy requirements for fliers so far as the status of the septum is concerned.

Dr. M. Martyn Kafka, of New York, cited his experience with test pilots at a commercial airplant, who ascend to high altitudes and frequently descend at a rapid rate. They complain of intense pain in the cheeks and forehead. In 1 instance a pilot who had an alveolar fistula was given relief by shrinking the mucosa about the opening. From this experience, Dr. Kafka concluded that it might be a good idea to shrink the nasal mucosa just before a particularly trying flight is undertaken.

A pilot with bronchial asthma who was cautioned against going above 18,000 feet (5,500 meters), reported having exceeded this altitude and, with the use of oxygen, having experienced no untoward effects. This fact was mentioned because of Army regulations which ground any man with bronchial asthma.

The speaker mentioned his own work with the pressure chamber at Columbia University and cited 1 instance of a man with a 4 plus deviation of the septum who was able to go as high as 47,000 feet (about 14,000 meters) without any unusual symptoms after preliminary shrinking of the mucosa with neosynephrine hydrochloride.

Dr. Rosenberg compared the effects of mass suction, frequently practiced in office treatment of sinusitis, with those described by the essayist. The results of Colonel Campbell's research would seem to offer a good argument against such a practice.

Dr. Samuel Salinger felt that Colonel Campbell's presentation was a clear exposition of a definite clinical entity. Considering predisposing causes, he mentioned the possibility of the size of the sinus having something to do with the incidence of hemorrhages such as the essayist described. The slides shown seemed to bear this out.

On comparing aerosinusitis with aerotitis, it is interesting to note that for the former there is lacking a simple prophylactic measure which is available for the latter, namely, autoventilation by the Valsalva pro-

cedure; excepted is vasoconstriction, which was mentioned.

In closing Colonel Campbell acknowledged the valuable assistance in this work which was made available by various physiologic units in the field, as well as the interest and help afforded by interested colleagues. Replying to Dr. Fenton's query, he had found the use of oxygen of little value in the prevention of this complication. Aerosinusitis is the result of a combination of factors which must operate in the right sequence to cause the condition described.

As to aerial flight aggravating sinusitis, the author was quite sure that this is true, and consequently persons known to have such a condition had better avoid flying.

Concerning the experiments at Northwestern University mentioned by Dr. Galloway, the essayist stated that people feel exhilarated at varying altitudes, and it might be possible that the therapeutic effects claimed were the result of the anoxia incident to altitude.

He did not believe that resection of the septum aided many of these patients. It would be indicated only when impingement of the septum on the turbinal causes blockage of the ostium.

Concerning the certainty of the diagnosis of hematoma, in his cases he is convinced that such was the condition, since it could be reproduced experimentally in dogs.

As to the relationship of the capacity of the sinus to the incidence of hemorrhage, the essayist agreed with Dr. Salinger: Aerosinusitis was observed most of the time in patients with large sinuses.

Summary of Some Facts Known Concerning the Common Cold. Presented by DR. A. C. HILDING, Duluth, Minn.

These facts about the cold seem to be fairly well established: It is a widespread contagious epidemic disease which occurs in all climates, especially in the autumn and the spring. It is spread by droplet infection and, like other communicable diseases, can be controlled by isolation. It is caused by a specific virus and is frequently followed immediately by complications due to bacterial infection. These are purulent rhinitis, otitis media, laryngitis and bronchitis. Chilling and exposure are definite etiologic factors, probably because of prolonged reduced temperature in the nasal mucosa. Immunity following attacks is brief as far as both the virus and the complicating bacteria are concerned. Vaccination is unsuccessful, apparently because both the virus and the bacteria have such poor antigenic properties. Prevention by the taking of vitamins or by so-called "hardening processes" are also largely unsuccessful.

The local pathologic process centers about an injury to the epithelium, presumably by direct action of the toxins on the epithelial cells, and consists in destruction of the superficial cells. There is marked submucosal cellular reaction. The secretion increases tremendously in volume and contains a lower percentage of mucin. There is at least some indication that the lysozyme content decreases even before the volume increases. The change in p_H is small, if any, and tends toward the alkaline side. Systemic pathologic changes are largely unknown.

Treatment is not very successful and consists principally in symptomatic measures and attempts to prevent complications.

The essayist showed a number of slides depicting the progressive changes in the nasal mucosa, beginning within a few hours after the onset of a cold and at various periods up to ten days. They revealed [illegible] edema in the submucosa, necrosis of epithelial [illegible] fragmentation, vacuolation, disarrangement of cell [illegible] mation and other changes, the result of toxic destruc[illegible]

DISCUSSION

Dr. George B. Wood agreed that a cold in its [illegible] stages is a virus infection and that it is urgent [illegible] one try to prevent the effects of secondary bacte[illegible] invasion. He demonstrated a practical method of [illegible] taining material from the nose for culture without [illegible] ger of contamination. Commenting on the que[illegible] as to the cause of the secondary bacterial activity, [illegible] Wood was not sure whether it was due to reduced [illegible] resistance or to some reaction between the virus [illegible] the bacteria. He recalled some experiments he [illegible] done on dogs some years ago at the University [illegible] Pennsylvania, in which infection of the sinuses c[illegible] be induced only after the mucosa had been mechani[illegible] irritated.

Dr. H. Marshall Taylor, of Jacksonville, Fla., m[illegible] tioned the effects of chilling without exercise and [illegible] exercise, the former being followed by leukop[illegible] whereas the latter is accompanied by leukocytosis. [illegible] would appear that exericse which increases respira[illegible] might be the cause of a rise in the temperature of [illegible] nasal mucosa, thus increasing its resistance to infec[illegible]

Dr. Samuel Salinger mentioned two items w[illegible] might have a bearing on the predisposition to c[illegible] one being a relatively dry mucosa and the other [illegible] native constitution. There is a difference in vasom[illegible] reactability between the sympathetic and the para-[illegible] pathetic types of persons, which can be determined [illegible] a variety of procedures.

Dr. Arthur W. Proetz, of St. Louis, asked whe[illegible] Dr. Hilding thought that persons susceptible to chi[illegible] were generally in the group with hypothyroidism. [illegible] there any connection between constantly feeling c[illegible] and susceptibility to chilling?

Dr. Hilding, in closing, cited the work of Paul [illegible] Freese in their studies of this problem up in Spitsber[illegible] Svalbard, Norwegian Arctic Territory. (Paul, J. H., [illegible] Freese, H. L.: An Epidemiological and Bacteriolog[illegible] Study of the Common Cold in an Isolated Arctic C[illegible] munity [Spitsbergen], *Am. J. Hyg.* **17**:517-535, 19[illegible] They found that the natives isolated from the world [illegible] seven months were free from colds until the first [illegible] came in the spring; then colds developed in 90 per [illegible] of them. A second round of colds developed in [illegible] summer, and then the incidence dwindled in the [illegible] after the shipping season closed. They interpreted [illegible] to indicate that the cold virus is short lived and [illegible] out in a community unless new carriers come in.

Dr. Hilding stated he could corroborate Dr. Wo[illegible] experiments relative to the importance of trauma a[illegible] factor in the development of infection.

As to exercise and the possible raising of the t[illegible] perature in the mucosa, he could not express an opi[illegible] although he did know that in the normal person exp[illegible] to chilling the depression of temperature did not [illegible] more than ten minutes.

He agreed that drying of the mucosa was a [illegible] disposing factor. He felt that there might be so[illegible] thing in the theory of sympathetic-parasympath[illegible] constitution as a factor, and would suggest it as a t[illegible] for further research.

As to the question of hypothyroidism in relation [illegible] susceptibility to colds, he was unable to express [illegible] authoritative opinion.

reoscopic vs. Single Roentgenograms. Pre-ented by DR. F. M. LAW, New York.

he information obtained from stereoscopic roent-grams of the sinuses is much more valuable to the eon than that obtained from a single roentgeno-n. It is much more satisfactory to look into a ty with the third dimension visible, on a stereoroent-gram, than to look at the cavity on a flat film. A of conditions in which this is especially true is n, as well as a reason for the necessity of such d information.

tereovision is not available to many surgeons, and apparatus is too expensive for the average private e. A method of fusing the vision without such ap-tus is described and illustrated. The value of the ography is briefly discussed; it is a too expensive hod for the average case of sinusitis, but it is indis-able in laryngologic work.

fter the presentation of the paper Dr. Law gave a tical demonstration as to how roentgenograms may iewed stereoscopically.

Morning Session

Thursday, June 8, 1944

yngocele. Presented by DR. H. MARSHALL TAY-LOR, Jacksonville, Fla.

he pathogenesis of laryngocele is ascribed to dilata-of the sacculus, which is a degenerated relic of sacs leading out of the ventricle in lower mammals. yngocele may be external, internal or both com-d. In the first-named type the mass finds exit to neck by pressure through the thyrohyoid membrane. cases have been reported in the literature.

he author's case was that of a man aged 35, whose ptoms dated back many years. A swelling of con-rable size would appear in the neck on singing or r exertion. Roentgenograms confirmed the diag-s.

he tumor was removed by external approach. There been no recurrence in two years.

DISCUSSION

r. Chevalier L. Jackson, of Philadelphia, stated that had never seen a laryngocele requiring external ration until recently. The laryngocele was in a g service man and could be demonstrated externally he neck but was easily reduced. Dr. Jackson ex-ed to operate on this patient in the near future and ed to present as successful a result as obtained by Taylor. He found planography very successful in onstrating the location and the extent of the sac his case.

r. Leroy Schall, of Boston, reported having seen 4 ngoceles at the Massachusetts Eye and Ear Infirm-during the past few years. One laryngocele was teral. He mentioned having reported a case recently hich a laryngocele was associated with a cancer of larynx; laryngectomy was performed. He had thus failed to find a similar case described in the litera-except a case reported by Dr. New in an article cysts of the larynx.

r. Schall also mentioned a patient who had an ex-al swelling over the larynx for years but refused ration until finally she came to the hospital in a atose condition. The autopsy specimen showed a bined internal and external laryngocele.

Dr. Samuel Iglauer, of Cincinnati, recalled a case of his, referred to by the essayist. The boy when first seen by him was about 8 years of age. Operation was not considered necessary at the time. He saw the patient twenty years later with apparently no increase in symptoms. The patient later entered the army, where he died of disease or injury. Apparently, in the speaker's opinion, unless a laryngocele produces symptoms there is no particular need for operation.

Dr. Iglauer felt that the term "laryngocele" was unfortunate, since the protrusion is really an aerocele. Commenting on Dr. Taylor's statement that the sac is a sort of accessory lung, such as that found in apes, and that when one of the apes closes his glottis he has a quantity of air trapped on which he could draw for oxygen, the speaker expressed the opinion that there could be little absorption of oxygen from a sac of that size. He suggested that in the animal this sac might possibly serve as a life preserver when in the water or, as he had read somewhere, could be blown up to scare possible enemies. He was unwilling, however, to express an opinion on the matter.

Concerning the statistics cited, Dr. Iglauer expressed the belief that the reference to the 85 cases collected by Moore was inaccurate, because Moore was dealing with cases of various other lesions of the larynx, among which there were only 10 or 12 cases of pure laryngocele.

As to diagnosis, Dr. Iglauer was satisfied with roentgenograms taken while the patient blew up the sac by doing the Valsalva act. The same effect can be obtained in a child by giving him a whistle to blow. He saw no objection to putting a needle into the sac for diagnosis, although if the sac did become infected, one would have a serious infection of the neck to deal with.

Dr. W. L. Simpson, of Memphis, Tenn., recalled his case, which Dr. Taylor mentioned, in which the laryngocele was of traumatic origin, the patient having been struck in the neck. At operation an opening was found in the left thyroid cartilage, the size of a small egg, through which the mass could be pressed back into the larynx and then reblown by the patient. He also mentioned the use of iodized poppyseed oil for more accurate delineation of the sac.

Dr. George B. Wood stated that, although he had personally never seen a laryngocele, he had always taught that there were two types, namely, the traumatic and the idiopathic. They are differentiated by the fact that in the former the air is in the connective tissue, with an inflammatory capsule about it, whereas in the idiopathic type the sac is lined with epithelium.

Dr. Charles Imperatori, of New York, mentioned having studied a case of this kind about thirty years ago, when their x-ray apparatus was either absent or nonfunctioning and the diagnosis was arrived at by palpation and auscultation. He felt that the maneuver whereby the larynx is moved from side to side, producing normally a crepitus, is one that has definite value and should be employed more frequently.

Dr. F. M. Law said that planograms or plain lateral roentgenograms are satisfactory means of revealing the mass which, being filled with air, is easily outlined in the dense tissues of the neck. Pictures should be taken on both inspiration and expiration.

Dr. Taylor, in closing, referred Dr. Iglauer to Negus' book on the mechanism of the larynx for an answer to his suggestions in physiology (Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929). As for the sac acting as a life saver in the ape, the theory was untenable because that ani-

mal is far from being aquatic, making no effort to swim when put into the water and drowning without effectual effort at saving himself.

Pathologic Alterations of Nasal Mucous Membrane and Suggestions as to Treatment. Presented by DR. LEROY A. SCHALL, Boston.

A basic knowledge of the histology, the pathology and the physiology of the nasal mucosa should predicate its treatment. This paper presents the accepted pathologic changes due to acute, turgescent, vasomotor and atrophic rhinitis. The highly controversial subject of the treatment of these conditions is likewise presented. An attempt is made to justify the treatment by the pathologic changes seen in each disease.

DISCUSSION

Dr. Arthur W. Proetz was in agreement with Dr. Schall and heartily seconded his suggestions as to treatment. Too much emphasis cannot be placed on the simple procedures which have stood the test of time and proved their reliability. It is always well that some one sound the note of warning against being misled by fancy therapeutic measures backed by high pressure propaganda.

Dr. Proetz is not in favor of nasal irrigation; he prefers capillary suction with a slender tube which enables one to draw secretions from the meatuses that cannot be reached by irrigation. He is opposed to mass suction with nostrils occluded. He offered a simple procedure for the relief of the dry nose, namely, a 5 per cent glycerin, 5 per cent alcohol solution in isotonic solution of sodium chloride. A few drops of perfumed spirit N. F. are added, and the solution is stained with sensitive yellow. It is stimulating, deodorant and effective when used three or four times daily, half a dropperful in each nostril. The alcohol induces local hyperemia and stimulates the flow of mucus. Dr. Proetz also has found this preparation useful in the bronchi of children with tracheobronchitis associated with thick secretion.

Dr. John H. Foster, of Houston, Texas, called attention to the use of ethylene disulfonate as reported in the *Journal of Pediatrics*. Its use is based on the stimulation of carbohydrate metabolism. The speaker and Dr. Cody had experimented with the use of dinitrous phenol, with excellent results in a number of cases but had given it up, fearing the drug might prove dangerous. His experience with operative narrowing of the nasal chambers in atrophic rhinitis had been favorable, and he was surprised that Dr. Schall had had no luck with the operation. Dr. Foster also mentioned good results from the prolonged use of cod liver oil in atrophic rhinitis.

Dr. Charles Imperatori reported his experience with a family of 4 girls, aged 7 to 14, and the parents, all suffering from atrophic rhinitis. Using estrogenic substance following irrigation, he noted that the degree of improvement was in inverse ratio to the age; that is, the greatest relief was in the youngest and the least in the oldest.

Dr. Thomas E. Carmody thought there might be some value in the modern methods of dress, which permit greater exposure of the skin. He agreed with Dr. Schall on the virtue of rest in bed and powder of ipecac and opium N. F. in acute conditions. He was not convinced, however, that the habit of sleeping in a cold room with windows wide open was beneficial. He favored the limited use of silver salts but warned against their prolonged use, having seen argyria resulting in some cases. He thought solutions of neosilvol da ous because the pale color is deceiving. Dr. Ca agreed with Dr. Proetz as to the usefulness of ca suction and was also opposed to routine irrigation.

Dr. Thomas Galloway expressed the belief tha should not be too hasty to condemn certain proce which in some hands have proved beneficial. By s ing the processes of inflammation and nature's m of overcoming them, one frequently finds certain sures useful which assist and hasten the natural Just as roentgen therapy heightens the intensity acute inflammation and hastens resolution, so judicious use of packs, hypertonic solution of magn sulfate and partially ionized silver salts may a plish the same thing in the nose.

Dr. Frederick Z. Havens, of Rochester, Minn., tioned a report of investigations of heating and ve tion which proved that in the average building was sufficient infiltration of air to provide a com interchange between inside and outside seven time hour. This fact should put the quietus on the air, open window fetish.

Dr. Schall, in closing, agreed as to the valu capillary or spot suction. Yet at certain times irrig and packs may be useful, provided the therapy overdone.

Replying to Dr. Foster, he stated that he had ha experience with ethylene disulfonate.

Inter-American Relations in Otolaryngol Presented by DR. CHEVALIER L. JACKSON, Phila phia.

Beginning in 1942, when the Committee on I American Relations in Otolaryngology was appo by Dr. Wherry with the speaker as its chairma bring all the countries of the Western Hemisphere closer communication for the exchange of ideas an promotion of mutual scientific interests, steady pro has been made. In 1943 a number of represent otolaryngologists from Central and South Am came to Chicago and participated in the comm meetings. At this time the Pan-American Congre Otolaryngology was proposed and financial arra ments were made. At the present time, contact is maintained with colleagues in the other countries. American societies of otolaryngologists have bee formed of the proposed congress. The Presider the South American Congress is being informed o plans and invited to bring that congress to this co in conjunction with the annual meeting of the Ame Academy of Ophthalmology and Otolaryngolog 1945, at which time the first meeting of the Pan-A can Congress of Otolaryngology will be held. I change of postgraduate students and residents be North and South America is being fostered and couraged.

A number of letters from South American colle were presented, indicating enthusiastic response t proposals of the committee.

Prior to the discussion of Dr. Jackson's paper, P dent Imperatori took occasion to introduce three vi colleagues from Latin America, who received a co welcome.

Dr. Ralph Fenton complimented Dr. Jackson o industry and care with which he and his committe worked on the plans for the proposed congress. felt that otolaryngologists in the United States much to gain from the experience of Latin Ame colleagues and that a free interchange of ideas be them should prove a great stimulus for all.

Imperatori agreed heartily with Dr. Fenton and ented on the utter lack of understanding that exists ny circles. Differences of temperament will prove stacle once personal contact is established. In hese differences will, in the long run, be found imaginary and fictional. The aims of these s are identical, and nothing but good can result closer collaboration.

Afternoon Session

Thursday, June 8, 1944

Problems in the Surgical Treatment of lateral Abductor Paralysis of the Larynx. resented by DR. JOSEPH D. KELLY, New York.

s paper deals with 34 operations for the relief ateral abductor paralysis of the larynx and some surgical problems encountered in the course of operations. Dr. Kelly describes the type of case the best result and the failures encountered in pting to do secondary operations at the sites of al operations. He also covers some changes in chnic of the original procedure and the reasons aking these changes.

Kelly showed several slides. Slide 1 showed the erative action of the constrictor pharyngis inferior e and the cricopharyngeus muscle causing a pull- of the right side of the larynx, due to removal rt of the right thyroid plate. This illustrates the tance of conserving every bit of the thyroid car- in order to insure an adequate airway.

de 2 showed the attachment of the stylopharyngeus e, which pulls on the posterior part of the thyroid ge. Collapse of the whole posterior soft tissues result if any of the thyroid cartilage is removed at oint.

de 3 also showed the importance of conserving d cartilage to prevent a falling of the tissues con- some of the intralaryngeal muscles, which have eir fixed anchor.

DISCUSSION

John H. Foster inquired whether it would be of tage in the healing for the patient to wear a rub- hold tube such as the Jacksons recommended for on of strictures.

J. W. McCall, of Cleveland, stressed the impor- of identifying and fixing the arytenoid cartilage. the relationship between the arytenoid cartilage he ala of the thyroid cartilage varies in different ns, it may not be so easy to determine just where ke the window. To overcome this difficulty, the er described how he uses a Jackson anterior oscope in a darkened room, by which means the oid cartilage can be located through the thyroid age and the exact point for the window outlined. the window is made, the laryngoscope is again uced, the interarytenoideus muscle is severed and rytenoid cartilage is mobilized by disarticulation the cricoid cartilage and pulled through the win- by means of a suture which is fixed to the peri- rium of the thyroid cartilage.

Mervin C. Meyerson, of New York, described a dure suggested by Dr. Imperatori which had d successful in 1 case. It consists of simple otissure, passage of a suture through the posterior f the vocal cord, carrying it through the cartilage ying it in the subcutaneous tissues external to the d cartilage. The procedure is simple and avoids the necessity of removing any cartilage. In another case the operation failed because of ensuing perichondritis.

Dr. Thomas C. Galloway felt that there was no need of removing the arytenoid cartilage when it can so easily be pulled into the window of the thyroid cartilage and fixed. He also agreed that the use of the laryngoscope is an advantage in locating the site for the window.

Dr. Francis L. Weille reported having followed a patient who had been operated on by Dr. Kelly. Being impressed with the fact that the patient did not aspirate fluids into the larynx because of the disturbed musculature, he asked Dr. Kelly for an expression on this point.

Dr. Kelly, in closing, stated that as a rule he is opposed to the use of foreign bodies in the larynx, such as the mold mentioned by Dr. Foster, on the ground that the less irritation after either an intralaryngeal or an extralaryngeal procedure the less possibility there is of the formation of scar tissue. In his experience the suture alone has been sufficient.

Concerning the question whether the arytenoid cartilage should or should not be removed, Dr. Kelly said he had no quarrel with those who advocate its retention, since he conceded there were different procedures that yielded good results in different hands. He recommends his operation to those who are not satisfied with their previous technic or who have no procedure to start with.

Concerning the ability to swallow liquids without aspiration, Dr. Kelly stated that it was astonishing to observe this phenomenon, which to some observers might seem to contradict some of Dr. Negus' theories on the mechanism of deglutition.

Extralaryngeal Surgical Approach for Arytenoidectomy: Bilateral Abductor Paralysis of the Larynx. Presented by DR. HENRY B. ORTON, Newark, N. J.

Laryngologists have recognized that surgical alteration of the neck, especially thyroidectomy, is one of the most frequent causes of bilateral abductor paralysis of the larynx.

Surgeons and laryngologists for some time past have been devising methods whereby patients with this type of paralysis could obtain relief from their condition. Today some form of arytenoidectomy with lateral fixation of the cord seems to be the operative procedure. Removing the posterior half of the ala of the thyroid cartilage gives adequate access to the arytenoid cartilage without opening the pharynx or the larynx. The first step in actual attack on the arytenoid cartilage itself must be proper exposure. The main points of the operation are as follows:

1. The lateral transthyroid approach gives access to a wider field of operation.

2. The longitudinal incision and splitting of the sternohyoid and thyrohyoid muscles is used instead of the transverse incision.

3. A direct identification of the arytenoid cartilage is made by palpation beneath the laryngopharyngeal aponeurosis.

4. The arytenoid cartilage is under complete vision during the entire arytenoidectomy.

5. A direct extralaryngeal surgical attack to the side of the larynx is possible.

DISCUSSION

Dr. Joseph D. Kelly stated that it was important not only to provide the patient with an adequate airway

but to see to it that he retained some sort of voice. He prefers to remove the arytenoid cartilage, since retention of this organ makes it necessary to separate the cords so far that the vocal function is definitely impaired. Removal of the arytenoid cartilage provides adequate breathing space without the necessity of dragging the vocal cord so far laterally as to prevent phonation. Dr. Kelly admitted that Dr. Orton's operation was simple and could be done without making a transverse incision or cutting the pretracheal muscles. However, removal of one third of the posterior part of the thyroid cartilage and sewing the cord out result in the formation of a great deal of scar tissue, which is likely to pull on the posterior part of the larynx, thus partially blocking that region. Dr. Kelly ascribes the success of his operation to preservation of the thyroid cartilage, which Dr. Orton removes in part.

Dr. Lawrence R. Boies, of Minneapolis, reported a successful result in a 37 year old woman, who was operated on by Orton's technic. The voice was not quite as good as previously, but the airway was satisfactory. He was impressed with the simplicity of the procedure.

Dr. Mervin C. Meyerson, amplifying his discussion of Dr. Kelly's paper, stated that the position of the vocal cord is not so much dependent on the entire arytenoid cartilage as on the vocal process. Therefore, his suggestion that the vocal process be severed through a laryngofissure and the posterior part of the cord pulled laterally by a suture passed through the thyroid cartilage should serve the purpose with minimum disturbance to function or removal of tissue.

Dr. Orton, in closing, admitted that the voice is not as good after these operations as before, but the same is true of almost any external operation on the larynx. As for the scar formation mentioned by Dr. Kelly, he had found that the tendency was to contract laterally rather than medially, provided the mucosa was not damaged.

CASE REPORTS

An Unusual Case of Carcinoma of the Larynx. Presented by Dr. Louis H. Clerf, Philadelphia.

Fibrosarcoma of the Larynx and Congenital Dermoid of the Pharynx. Presented by Dr. John H. Foster, Houston, Texas.

Neurofibroma of the Nasopharynx. Presented by Dr. Frederick T. Hill, Waterville, Maine.

DISCUSSION

Dr. John H. Foster mentioned a case similar to Dr. Hill's which he had reported several years ago. He commented on the ease with which the tumor shelled out and the comparative absence of bleeding. Horner's syndrome was present for a few days after the operation.

Dr. Andrew A. Eggston, of New York, reported his observations in a case in which Dr. Negus operated about a year ago. The tumor was located on the left side behind the tonsillar pillar and was responsible for paralysis of the recurrent laryngeal nerve. It shelled out easily, and, as in Dr. Foster's case, its removal was followed by Horner's syndrome.

Dr. Eggston showed several slides illustrating the histopathology of the growth.

Dr. R. S. Moore mentioned a similar tumor which he removed from the larynx about fifteen years ago. There has been no recurrence to date.

Atresia of the Pharynx Following Tonsillec and Adenoidectomy. Presented by Dr. Cu Imperatori, New York.

DISCUSSION

Dr. Thomas E. Carmody mentioned a proced which a flap from the cheek or the oral side palate is turned under to cover the raw surface the palate has been released. This procedure, ho does not cover the raw area on the posterior w the pharynx. In a case in which he expected to soon, Dr. Carmody planned to use a split graft.

Dr. Gordon Berry, of Worcester, Mass., state he was under the impression that the procedure de by Dr. Imperatori had been described by Dr. Ro Rochester, Minn., before Dr. MacKenty.

Dr. Imperatori, in closing, stated that he clai originality for the operation but had been und impression that it was first described by MacKe

Congenital Lymphangiomatous Macroglossia Cystic Hygroma of the Neck. Presen Dr. D. M. Lierle, Iowa City.

Several slides were shown illustrating the appe of the tongue at various stages, as well as the pathology of the growth.

DISCUSSION

Dr. Charles Imperatori recalled a similar case reported before this society in 1940 or 1941, in the child suffered so much from dyspnea that th way it could breathe was with its head over t of a pillow, similar to the Rose position. In thi the growth was treated by aspiration and inject a sclerosing fluid. The patient recovered.

Malignant Recurring Nasal Hemorrhage o determined Cause. Presented by Dr. S Salinger, Chicago.

Several biopsy slides were shown, as well as graphs of the external lesion. There was no disc

Muscles of Phonation and Deglutition as Vi Through an Operative Wound in the F and Ethmoid Sinuses. Presented by Dr. Simpson, Memphis, Tenn.

A motion picture with running commentary speaker comprised the greater part of the presen

Tumor of the Carotid Body. Presented by Du Viole, Los Angeles.

DISCUSSION

Dr. Walter B. Hoover, of Boston, stated that recently seen 2 cases closely resembling that de by Dr. Viole. One patient, a man of 30, was unsu fully operated on by both the external and the i approach. The other patient, a woman of 50, tumor presenting in the pharynx behind the which was pushed forward, and extending up base of the skull; it also presented at the angle jaw. In this case the common carotid artery wa pressed for several days prior to operation so could be tied if necessary. However, the tum removed without this being done.

In the first case the tumor was removed exter a second operation. It was found to be highly va In both cases tracheotomy and use of a feedi were necessary for a short time.

Book Reviews

pacio perilimfatico otico. By Dr. Mario Rius, ntevideo, Uruguay: Editorial Mosca Hermanos, ., 1943.

e Perilymphatic Space of the Ear" is a volume pages, liberally illustrated, devoted to the em- gy, the anatomy, the physiology, the pathology e therapy of the labyrinth of the inner ear. The s well printed on high grade paper, with a com- table of contents and an alphabetic author bibli- y. Lacking only is an alphabetic index of the t matter, which might have added somewhat to cessibility of the material. Especially noteworthy e series of photomicrographs of histologic sec- made through the labyrinth, all carefully labeled, he excellent drawings of gross sections made h the labyrinth, carefully labeled.

subject matter is treated completely and with critical judgment. The comparative anatomy of byrinth is well illustrated. The physiology in- theories of the mechanism of hearing. The rs devoted to diseases of the labyrinth and their ent are especially interesting. Ménière's disease arded as hydrops of the endolymphatic labyrinth, indicating that the author is well up to date in thology. Two photomicrographs from Lindsay produced to illustrate this condition.

surgical treatment of otosclerosis receives more 00 pages. The historical development of the ation operation and of the other attempt to im- otosclerosis by surgical means is well covered. ublished results of all of these procedures are d, with interesting and excellent editorial com-

author presents his own fenestration technic, a cation of Lempert's nov-ovalis technic, the chief nces from Lempert's technic being:

he operative cavity is confined to the aditus ad n and the attic without further opening of the d.

or irrigation the author uses Ringer's solution h liter of which has been added 100 cc. of plasma ke the albumin content equal to that of normal ph, with the further addition of 15 cc. of 5 per odium sulfathiazole.

binocular microscope is used in making the

small ivory implement is being experimented s a means to prevent closure of the fistula, but o soon to present the results.

cially commendable is the author's exceptionally te report of the cases in which he has operated, preoperative and postoperative audiograms on he ear operated on and the ear not operated on cases, the bad results with the good. A total patients were operated on, 1 by a modified lle-Lempert technic, the other 15 by the nov- technic of Lempert with the author's modifica- Of 10 cases in which there were good indications e operation, the fistula closed in 4; in 3 the results are too recent to permit him to judge. In 3 other cases there are persistent hearing improvements as follows: 21 decibels for the speech frequencies after ten months in 1 case, 11.6 decibels after nine months in 1 case and 15 decibels after nine months in 1 case.

This well written, well illustrated and well printed book can be highly recommended as a reference work on the labyrinth, particularly on all types of surgical treatment of the labyrinth.

Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E., Surgeon to the Royal Infirmary, Edinburgh. Third edition. A William Wood Book. Price, $4.50. Pp. 459. Baltimore: Williams & Wilkins Company, 1944.

The third edition of this text is well written and well indexed and consists of 459 pages, with 82 figures and 8 colored plates. The entire subject of otolaryngology and some phases of bronchoesophagology are covered in a comprehensive manner. There is an appendix on the use of sulfonamide compounds in diseases of the nose, the throat and the ear, including the indications, the control of dosage and the complications. Five pages of formulas are given at the end of the book.

It is recommended as collateral reading for students and practitioners. Graduate students in otolaryngology and specialists, however, are apt to find it somewhat elementary.

News and Comment

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1945 for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the secretary, Dr. Arthur W. Proetz, 1010 Beaumont Building, St. Louis 8, before March 1, 1945.

AMERICAN NEUROSURGEONS INVITED TO THE FIRST SOUTH AMERICAN CONGRESS OF NEUROSURGEONS

The First South American Congress of Neurosurgery will be held at Montevideo, Uruguay, March 1 to 4, 1945. An invitation to attend the Congress has been officially extended to North American neurosurgeons by the president, Prof. Dr. Alejandro H. Schroeder. Prof. Agdo. Dr. Román Arana Iñignes, Montevideo, Convencion 1287, is General Secretary of the Congress.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.

In compliance with the request of the Office of Defense Transportation and in the interest of the national war effort a meeting will not be held in 1945.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.

President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.

Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland.

Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Roche Minn.

Secretary: Dr. Arthur W. Proetz, Beaumont H St. Louis.

Place: Drake Hotel, Chicago. Time: May 30-31, 1

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University H pital, Ann Arbor, Mich.

Secretary: Dr. C. Stewart Nash, 708 Medical A Bldg., Rochester 7, N. Y.

Place: Drake Hotel, Chicago. Time: May 28-29, 1

SECTIONS:

Eastern.—Chairman: Dr. Oram R. Kline, 414 Co St., Camden, N. J.

Southern.—Chairman: Dr. Verling K. Hart, 106 7th St., Charlotte, N. C.

Middle.—Chairman: Dr. Carl H. McCaskey, 608 Gu anty Bldg., Indianapolis.

Western.—Chairman: Dr. Aubrey G. Rawlins, 384 P St., San Francisco.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., W cester, Mass.

Secretary: Dr. Isidore Friesner, 101 E 73d St., N York.

Place: Drake Hotel, Chicago. Time: June 1-2, 1

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

THE ARCHIVES OF OTOLARYNGOLOGY is published by the American Medical Association to stimulate research in the field of otology and laryngology and to disseminate knowledge in this department of medicine.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. George M. Coates, Chief Editor, 1721 Pine Street, Philadelphia, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago 10.

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Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

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of the American Medical Association

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ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original articles and general reviews in the field of pathology. Illustrated. Annual subscription price (two volumes): domestic, $6.00; Canadian, $6.40; foreign, $7.00. Single copies, 75 cents, except special issues.

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QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: domestic, $12.00; Canadian, $14.00; foreign, $14.00.

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